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*Schneeberg, N. G.; Perczek, L.; Nodine, J. H., and Perloff, W. H.: Methallenestril, a New Synthetic Estrogen, J.A.M.A. *161*:1062 (July 14) 1956.

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*Hodgkinson, C. P.; Igna, E. J., & Bukeavich, A. P.: *Ann. New York Acad. Sc.* 71:753, 1958.

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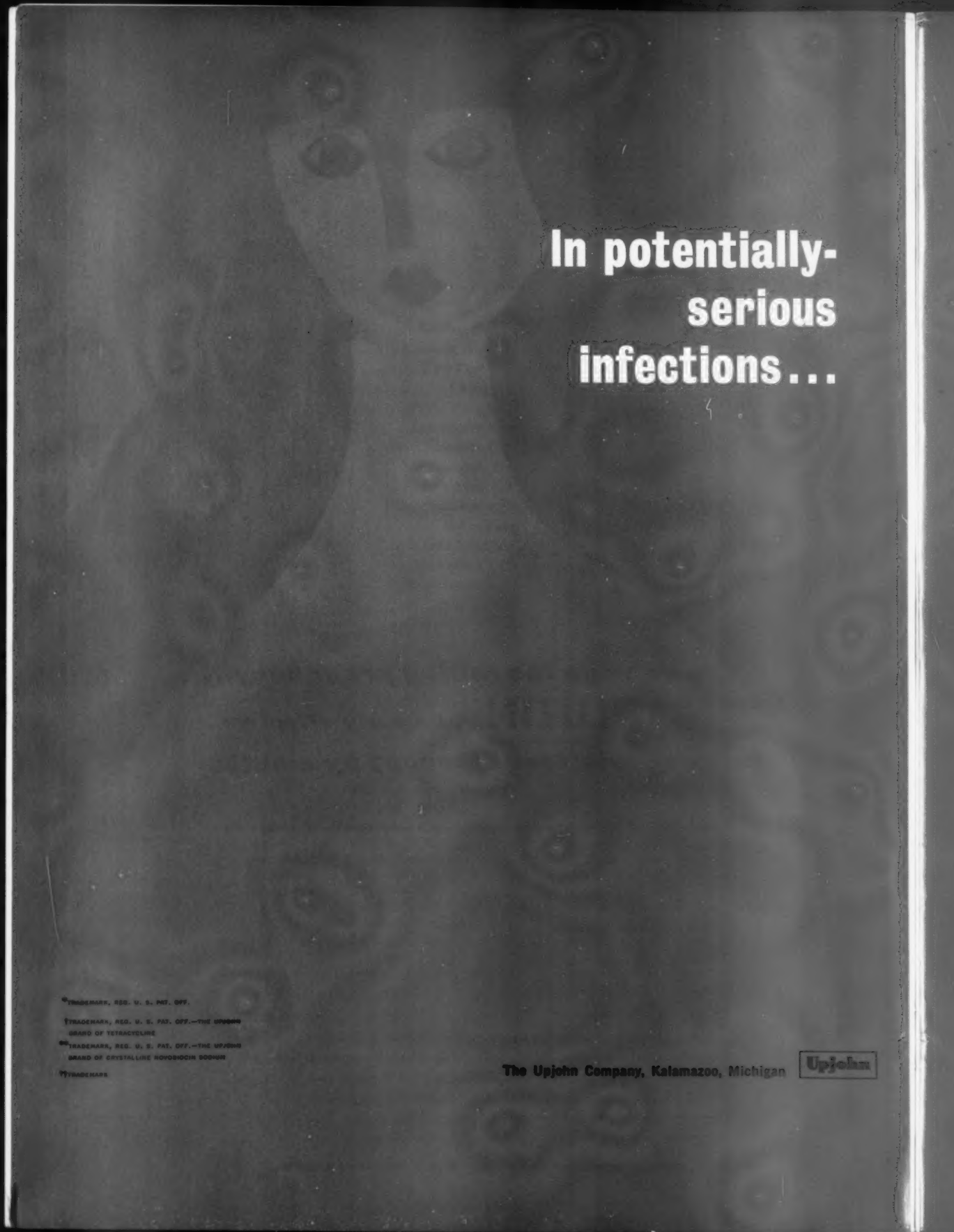

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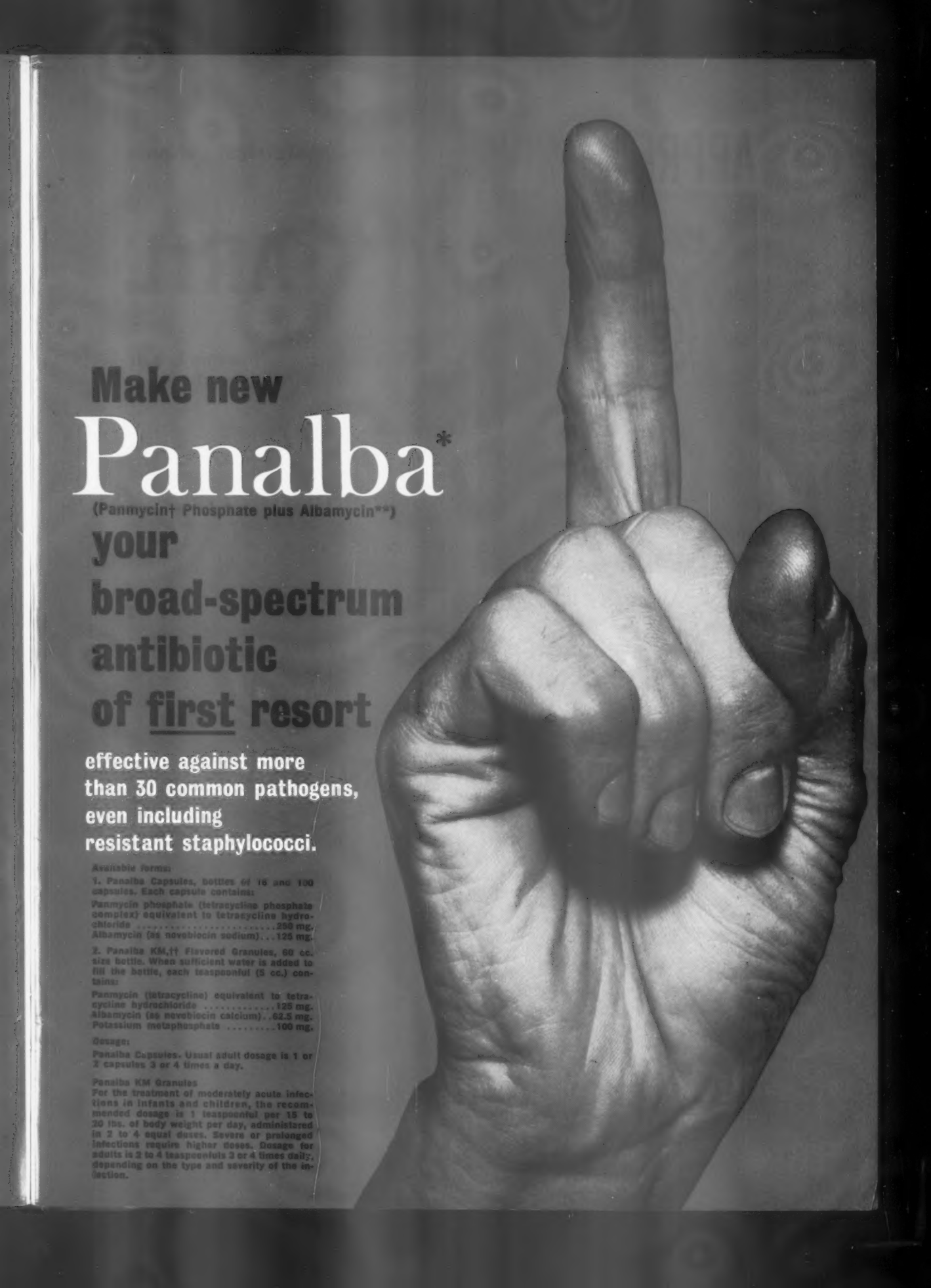
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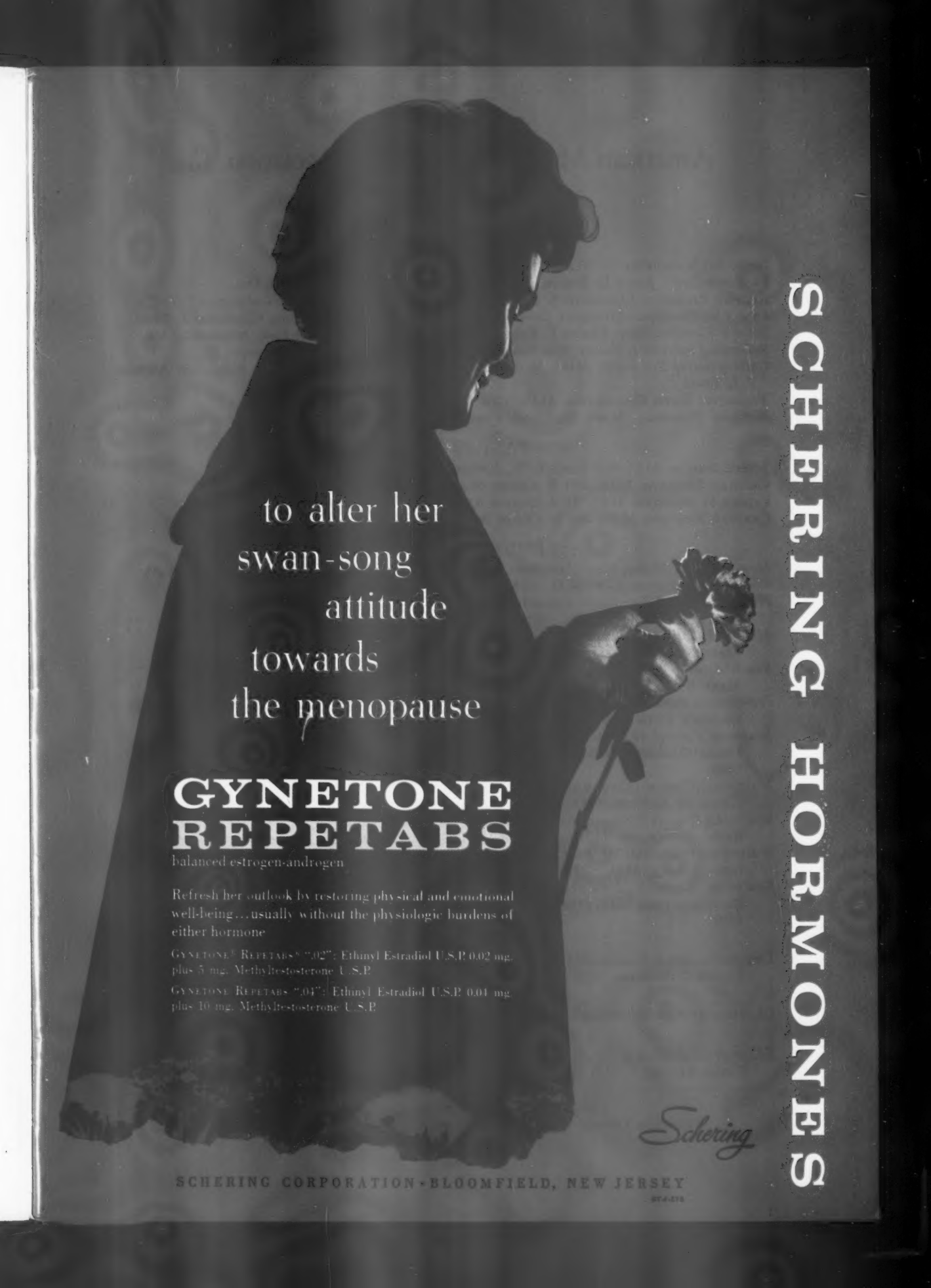
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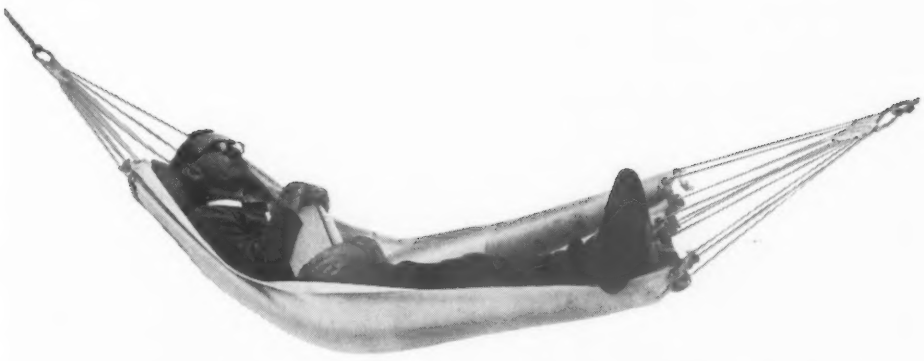

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(Continued on page 199)

Cor-Tyzine®

NASAL DECONGESTANT
ANTI-INFLAMMATORY
ANTI-ALLERGIC

tetrahydrozoline hydrochloride – prednisolone

Nasal Solution, 1/2-oz. dropper bottle, 0.1%. Each cc. contains 1 mg. tetrahydrozoline hydrochloride and 0.2 mg. prednisolone.

Pediatric Nasal Drops, 1/2-oz. bottle, 0.05%, with calibrated dropper. Each cc. contains 0.5 mg. tetrahydrozoline hydrochloride and 0.2 mg. prednisolone.

Note: As with certain other widely used nasal decongestants, overdosage may cause drowsiness or deep sleep in infants and young children. Use only as directed. Do not use Adult 1/4 oz. Use Pediatric Nasal Drops (0.05%) for children under six years. When Tyzine Nasal Spray is administered, it should be held only in an upright position.

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Science for the world's well-being

PFIZER LABORATORIES, Division, Chas. Pfizer & Co., Inc.
Brooklyn 6, N. Y.

NEW PRODUCT ANNOUNCEMENT

relieves depression during pregnancy and postpartum

acts promptly—controls the syndrome of mental depression *without* C.N.S. excitation; reduces depressive rumination and crying; restores natural sleep without barbiturate-like hangover.

significantly different—unlike amine-oxidase inhibiting energizers, Deprol produces no liver toxicity and does not adversely affect blood pressure; unlike C.N.S. stimulants, Deprol has no depressive aftereffects, does not cause insomnia or depress appetite.

Deprol†

...ge: Usual starting ...dose: 1 tablet q.i.d. When ...ecessary, this dose may ...be gradually increased up ...to 3 tablets q.i.d.

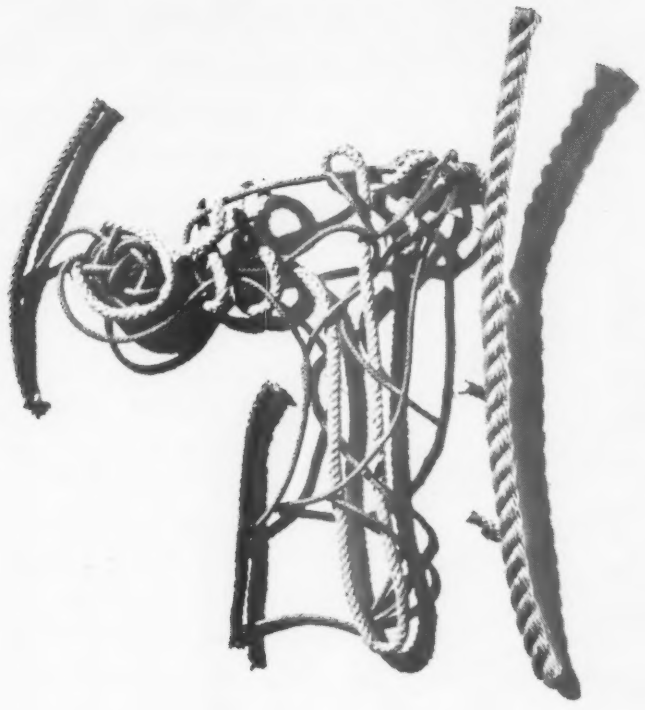
...positions: Each tablet ...contains 400 mg. meprobamate and 1 mg. 2-diethylaminoethyl benzilate hydrochloride (benactyzine HCl).

Supplied: Bottles of 50 scored tablets.

1. Current personal communications; in the files of Wallace Laboratories. 2. Alexander, L.: Chemotherapy of depression—Use of meprobamate combined with benactyzine (2-diethylaminoethyl benzilate) hydrochloride. J.A.M.A. 166:1019, March 1, 1958.

WALL... LABORATORIES, *New Brunswick, N. J.* *Literature and samples on request*

†TRADE-MARK



FURADANTIN®

brand of nitrofurantoin

PYELONEPHRITIS

"A DISEASE OF THE TUBULES"[1] as well as the glomeruli.

In pyelonephritis, "the tubules suffer from damage to their lining cells which show cloudy swelling, granular degeneration and diminution in size. Inflammatory cells and colloid casts are found in the lumen of the tubules. . . . The glomeruli remain normal over a long period."[1]

in addition to simple glomerular filtration, FURADANTIN is actively excreted by the tubule cells.

FURADANTIN "may be unique as a wide-spectrum antimicrobial agent that is bactericidal, relatively nontoxic, and does not invoke resistant mutants."[2]

Available as Tablets, Oral Suspension

References: 1. Smith, I. M., and Lenyo, L.: Am. Practitioner 9:78, 1958. 2. Waisbren, B. A., and Crowley, W.: A.M.A. Arch. Int. M. 95:653, 1955.

NITROFURANS—a new class of antimicrobials—neither antibiotics nor sulfonamides

EATON LABORATORIES, NORWICH, NEW YORK

in skin disorders

Decadron*

DEXAMETHASONE

treats more patients more effectively
a new order of magnitude in corticosteroid effectiveness
a new order of magnitude in margin of safety

Striking clinical results with DECADRON are reported† in 92 percent of 319 patients with dermatological disorders, including cases previously unresponsive or resistant to corticosteroids. There were no major complications, and even minor side effects occurred in less than eight percent of patients.

Moreover, in many cases reactions induced by previous steroid therapy, such as edema, Cushingoid appearance, headache, vertigo, muscular weakness, depression, hirsutism, and glycosuria, **disappeared during therapy with DECADRON.** †Analysis of clinical reports.

Dosage: One 0.75 mg. tablet of DECADRON will usually replace one 4 mg. tablet of methylprednisolone or triamcinolone, one 5 mg. tablet of prednisone or prednisolone, one 20 mg. tablet of hydrocortisone, or one 25 mg. tablet of cortisone.

Supplied: As 0.75 mg. and 0.5 mg. scored, pentagon-shaped tablets in bottles of 100 and 1000.

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American Medical Women's Association, Inc.

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(Continued on page 255)

Neocor-Tyzine*

tetrahydrozoline hydrochloride – prednisolone – neomycin

NASAL DECONGESTANT · ANTI-INFLAMMATORY · ANTI-ALLERGIC · ANTI-INFECTIVE

Nasal Solution, 1/2-oz. dropper bottle, 0.1%. Each cc. contains 1 mg. tetrahydrozoline hydrochloride, 0.2 mg. prednisolone, and 0.6 mg. neomycin (as sulfate).

Pediatric Nasal Drops, 1/2-oz. bottle, 0.05%, with calibrated dropper. Each cc. contains 0.5 mg. tetrahydrozoline hydrochloride, 0.2 mg. prednisolone, and 0.6 mg. neomycin (as sulfate).

Note: As with certain other widely used nasal decongestants, overdosage or continuous or deep sleep in infants and young children; KEEP OUT OF REACH OF CHILDREN OF ALL AGES. Use Pediatric Nasal Drops (0.05%) for children under six years. When Tyzine Nasal Spray is administered, it should be held only in an upright position.

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Eradicate Infection and Restore Normal Vaginal Flora...

New-'Neosporin'® brand Vaginal Suppositories

Trichomonacidal · Bactericidal

Each suppository contains:

'Aerosporin'® brand Polymyxin B Sulfate	20,000 Units
Neomycin Sulfate	5 mg.
Acetarsone	250 mg.
Beta-lactose	q.s. (approx. 1 Gm.)

In a base containing a surface active agent
Foil-wrapped—Box of 12

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The hyper-anxious hypertensive needs greater central control **Serpasil®**

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Hypertensive patients whose emotional disturbances cannot be controlled by agents which have no autonomic action need Serpasil: it acts on the central autonomic mechanisms which control reactions to stress. Thus, Serpasil not only lowers blood pressure, but eases the anxiety and agitation that contribute to your patient's hypertension.

DOSAGE: In the average patient not receiving other antihypertensive agents, the average initial dosage is two 0.25-mg. tablets daily, with a range of 0.1 to 1 mg. Later the Serpasil dosage should be reduced to 0.25 mg. or less daily for maintenance. SUPPLIED: *Tablets*, 0.1 mg., 0.25 mg., 1 mg., 2 mg. and 4 mg.

2/2638 MB

CIBA
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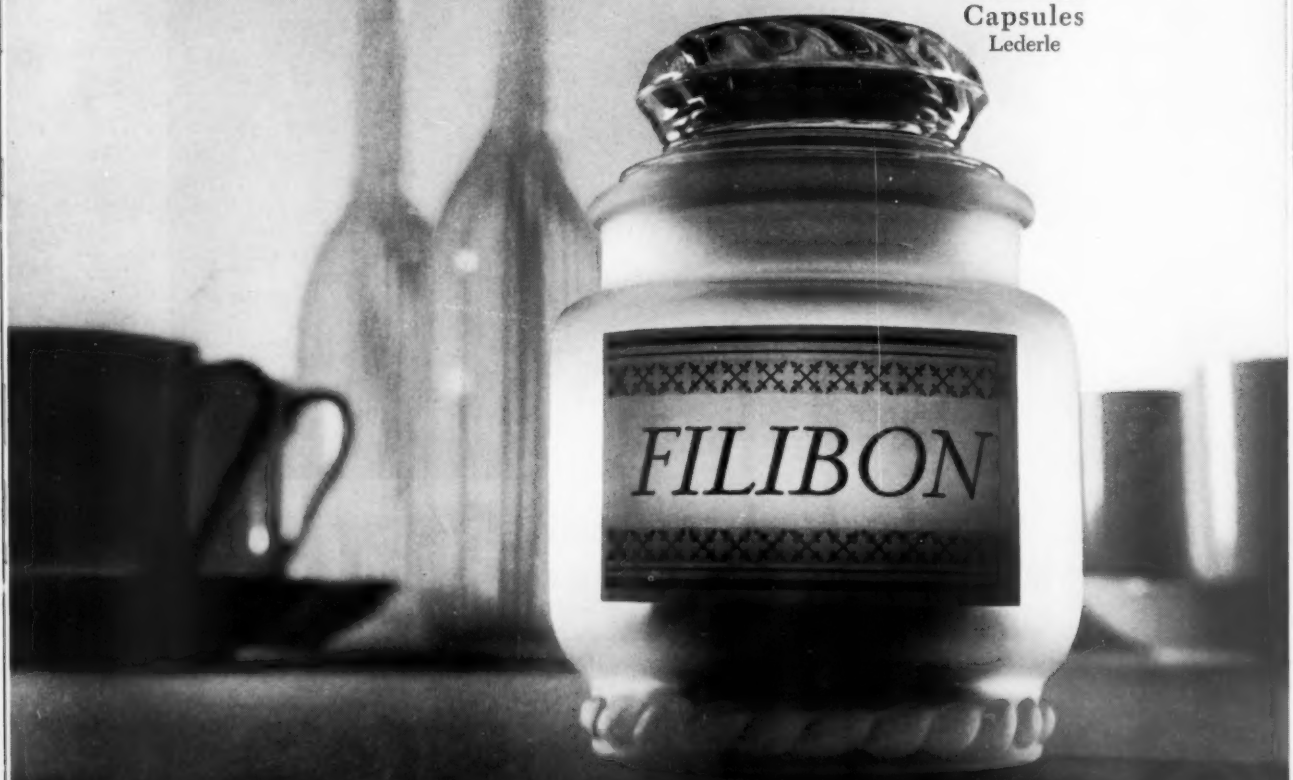
The attractive FILIBON jar reminds your pregnant patient to take her prenatal supplement daily. You can be sure that the vitamins, minerals, trace elements in this complete formula will provide the *everyday* nutritional support you prescribed.

And the patient feels better on FILIBON. Well tolerated ferrous fumarate and smaller, dry-filled capsules do not compound her problems with nausea of pregnancy.

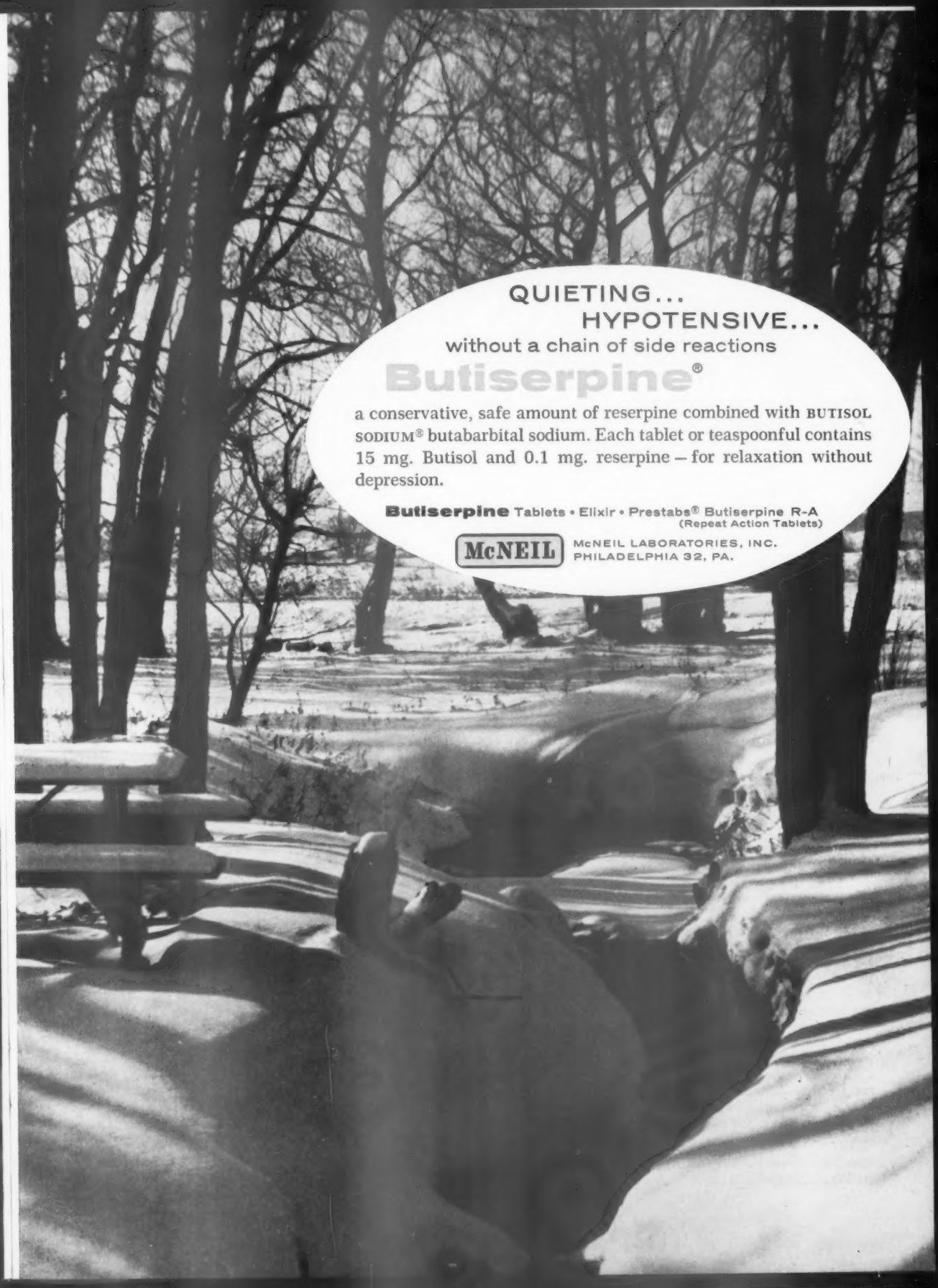
The up-to-the-minute formulation includes both vitamin K and AUTRINIC* Intrinsic Factor Concentrate, always enhancing B_{12} serum levels.

For complete formula see PDR (Physicians' Desk Reference) page 688.

Phosphorus-free
FILIBON*
Prenatal Capsules
Lederle



Lederle **LEDERLE LABORATORIES,** a Division of AMERICAN CYANAMID COMPANY
Pearl River, New York *Reg. U. S. Pat. Off.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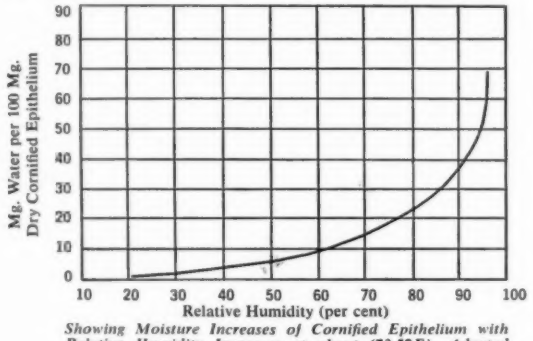
This is an advertisement.

the "MOISTURE FACTOR" in cosmetic care of the skin

Advanced Research for Improved Methods and Preparations—"The need for increasing the scope and number of investigations relating to fundamental problems of skin physiology, biochemistry, pharmacology, and toxicology cannot be stressed too strongly."[1] For example, emollients in many forms and formulations are in extensive use today to help meet the ever-increasing problem of dry skin encountered in modern life. Yet "...confusion exists...as to just what constitutes an emollient"[2] and how it functions physically and physiologically.

The "Moisture Factor" in Simple Skin Dryness—When skin lacks sufficient moisture in the horny layers, it is described as "dry." Symptoms range from mild superficial scaling to extremes of severe scaling, fissuring, itching, and inflammation.[2] Water evaporates from the surface more quickly than it is supplied from underlying tissues.[3]

Natural Surface Film Poor Barrier Against Water Loss Contrary to general belief, the natural surface lipid film is not an effective barrier against water loss to the atmosphere. Upon its removal with fat solvents, no increased evaporation rate is noted.[4] The water barrier probably lies in the deeper layers between cornified and non-cornified epidermis,[5] and does not prevent dehydration of the stratum corneum under most atmospheric conditions.[3] Prolonged exposures to temperature extremes, to the burning sun's rays or to strong wind, increase tendencies toward severe drying. Relative humidity is a critical factor—at values of 60 per cent or above, an equilibrium of water exchange exists which does not permit the moisture content of the horny layers to drop below 10 mg. water to 100 mg. of dry keratin.[3] At this value, the skin retains its pliability.[3]



Showing Moisture Increases of Cornified Epithelium with Relative Humidity Increases at about (73.5°F.). Adapted from Blank.[2]

Simple Emollients are Primarily Surface Acting—Dramatic evidence that "...water is the only known plasticizer for keratinized tissue"[2] has been presented. Dried out—but not defatted—skin sections become very hard and brittle. Prolonged contact with lanolin, olive oil or petrolatum does not restore their flexibility. But if allowed to absorb a little moisture, similar sections become soft and pliable.[2,3] "It is apparent, therefore, that the water content of the cornified epithelium is a more important factor in maintaining the flexibility of this layer than is its oil content."[2] *It can be assumed then that a simple emollient does not penetrate—it acts primarily on the surface. Its true effectiveness in the treatment of simple skin dryness depends on the help it can give the stratum corneum to maintain an adequate water content.*[3]

The "Moisture Factor" in Aging Female Skin—Consideration of the "moisture factor" in the care of aging skin becomes of increasing importance because of the deep-seated atrophic dermal changes which take place with aging. The epidermis becomes thinner and the outermost layers appear looser.[6] Mitotic activity is reduced in the basal layers and the deep epidermal cells begin to vacuolize.[6] Papillae gradually lose their waviness,[6] collagen fibers become thinner and elastin structure is rendered increasingly amorphous.[7] As a result, the corium becomes structurally less firm and elastic.[7] In aging patients loss of the capacity of the skin for hydration refers to the whole skin. "This occurs in the dermis due to the reduced hydration capacity of the collagen and ground substance..." as well as the horny layers.[8] It is apparent that simple surface-acting emollients cannot prevent or reverse dryness and other skin changes associated with aging.

Topical Estrogens and the "Moisture Factor" in Aging Skins—Because aging skin is prominently linked with waning ovarian hormones, topical estrogens, which "...penetrate the intact skin rapidly and with great ease,"[9] have been added to simple emollients in the attempt to arrest the degenerative process. Clinical reports[10,11,12,13] attest to the efficacy of topical estrogens in providing favorable response—structurally and functionally—on aging female skins. Prominently noted in these controlled studies were marked improvements relating to the "moisture factor." Increased succulence of the epidermal cells[10] and the derma[11] and enhanced ability to absorb fluids[11] have been reported. Estrogen-treated sites showed an increased water content[12] and it has been stated that "...there is definite support for the anti-wrinkling effect...based upon (a) the thickening of the epidermis, (b) plumping of the collagen fibres."[13] Hormone potencies used in these studies have been clinically established to be "...entirely safe"[14] and free of side effects.[15]

Beauty Through Science—In the Clinical Research Division of Helena Rubinstein, the application of established dermatologic principles to scientific cosmetology has succeeded in developing emollients and other cosmetic preparations that set the standards for the industry. Pioneering in cosmetic hormone therapy, this group has for years supported dermatologic, endocrinologic and cosmetologic studies to evaluate the benefits of topical hormones on aging female skin. Of special importance has been the recent development of Helena Rubinstein's Ultra Feminine Face Cream which combines natural estrogens with progesterone in concentrations clinically proved to be effective and safe. The dermatologic action of Ultra Feminine appears to be enhanced by the synergistic action of the *two* hormones. Through its estrogen content, Ultra Feminine favorably influences the vitally important "moisture factor" throughout the skin structure. Its progesterone content, through reactivation of impaired sebaceous function, increases surface emolliency and in addition helps to guard against undue moisture loss to the environment. A true product of scientific cosmetology, Ultra Feminine can help the patient maintain her youthful appearance well past "middle age."

References: (1) Lehman, A. J.: J.A.M.A. *164*:416 (May 25) 1957. (2) Blank, I. H.: J.A.M.A. *164*:412 (May 25) 1957. (3) Blank, I. H.: J. Invest. Dermat. *18*:433, 1952. (4) Winsor, T., and Burch, G. E.: Arch. Int. Med. *74*:428, 1944. (5) Rothman, S.: Physiology and Biochemistry of the Skin, Chicago, University of Chicago Press, 1954, p. 32. (6) Traub, E. F., and Spoor, H. J.: J. Am. Geriatrics Soc. *1*:805, 1953. (7) Nicholas, L.: J. M. Soc. New Jersey *54*:524, 1957. (8) Sulzberger, M. B.: Panel Discussion, Clinical Management of Skin Disease in Geriatric Patients, J. Am. Geriatrics Soc. *6*:575, 1958. (9) Rothman, S.: *loc. cit.*, p. 41. (10) Curth, cited in Peck, S. M., and Klarmann, E. G.: Practitioner *173*:159, 1954. (11) Traub, cited in Peck, S. M., and Klarmann, E. G.: Practitioner *173*:159, 1954. (12) Goldzieher, M. A.: Geriatrics *1*:226, 1946. (13) Peck, S. M., and Klarmann, E. G.: Practitioner *173*:159, 1954. (14) Sulzberger, cited in Peck, S. M., and Klarmann, E. G.: Practitioner *173*:159, 1954. (15) Goldzieher, M. A.: J. Gerontol. *1*:196, 1946.

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CLINICAL RESEARCH DIVISION
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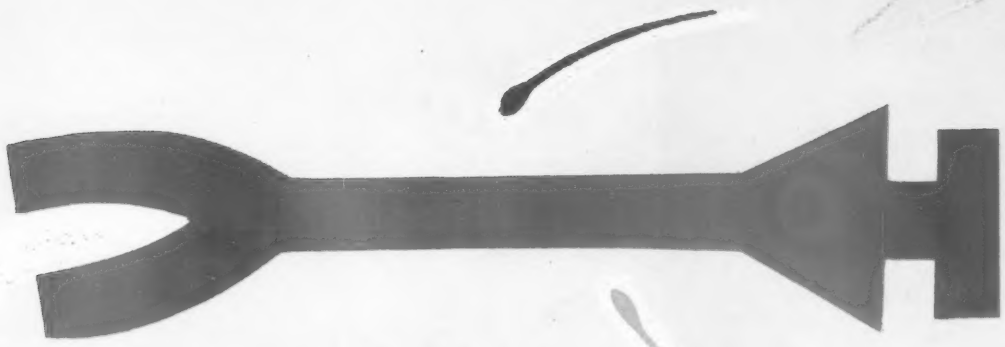

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your most fastidious patients

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"...is highly spermicidal....Its relative simplicity makes it very acceptable to the patient."*

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Composition: Nonylphenoxypolyethoxyethanol 5% in an oil-in-water emulsion at pH 4.5.

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means help

- For those who eat too much
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'Methedrine' dispels abnormal craving for food, subtly elevates the mood.

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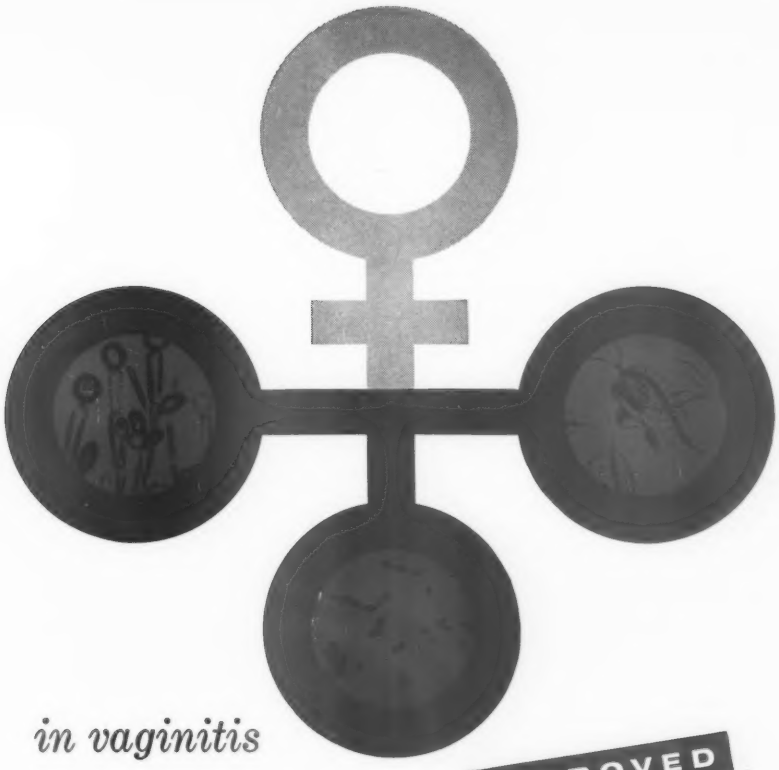

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IMPROVED

TRICOFURON®

destroys all 3 *principal pathogens*

Whether vaginitis is caused by Trichomonas, Monilia or Hemophilus vaginalis—alone or combined—TRICOFURON IMPROVED swiftly relieves symptoms and malodor, and achieves a truly high percentage of cultural cures, frequently in 1 menstrual cycle. TRICOFURON IMPROVED provides: a *new* specific moniliacide MICOFUR® brand of nifuroxime, the *established* specific trichomonacide FUROXONE® brand of furazolidone and the *combined* actions of both against Hemophilus vaginalis.

1. *Office* insufflation once weekly of the Powder (MICOFUR [*anti*-5-nitro-2-furaldoxime] 0.5% and FUROXONE 0.1% in an acidic water-soluble powder base). **2.** Continued *home* use twice daily, with the Suppositories (MICOFUR 0.375% and FUROXONE 0.25% in a water-miscible base).

℞ NEW BOX OF 24 SUPPOSITORIES WITH APPLICATOR FOR MORE PRACTICAL AND ECONOMICAL THERAPY.

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the trend is toward the NEW *Koro-Flex*

(CONTOURING)
DIAPHRAGM *worth investigation*

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KORO-FLEX (contouring) Diaphragm acceptable, not only where ordinary coil-spring diaphragms are indicated but for Flat rim (Mensinga) type as well.

Suggest the convenient-economical KORO-FLEX COMPACT 60-95 mm

Feminine Clutch-style bag with zipper closure. Diaphragm, tube KOROMEX Jelly (3 oz.) Cream (1 oz. trial size).

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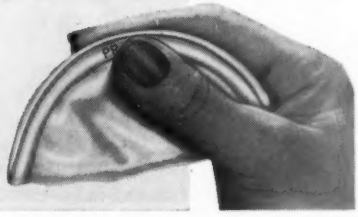
CONTOURING SPRING (ARCING TYPE)



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for her most personal cleansing

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1959 ANNUAL MEETING

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____Twin bedroom with bath ($14.00 per day)

I will arrive at _____a.m._____p.m. Date _____

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Sheraton-Ritz Carlton Hotel, Atlantic City, N.J. *June 4–7, 1959*

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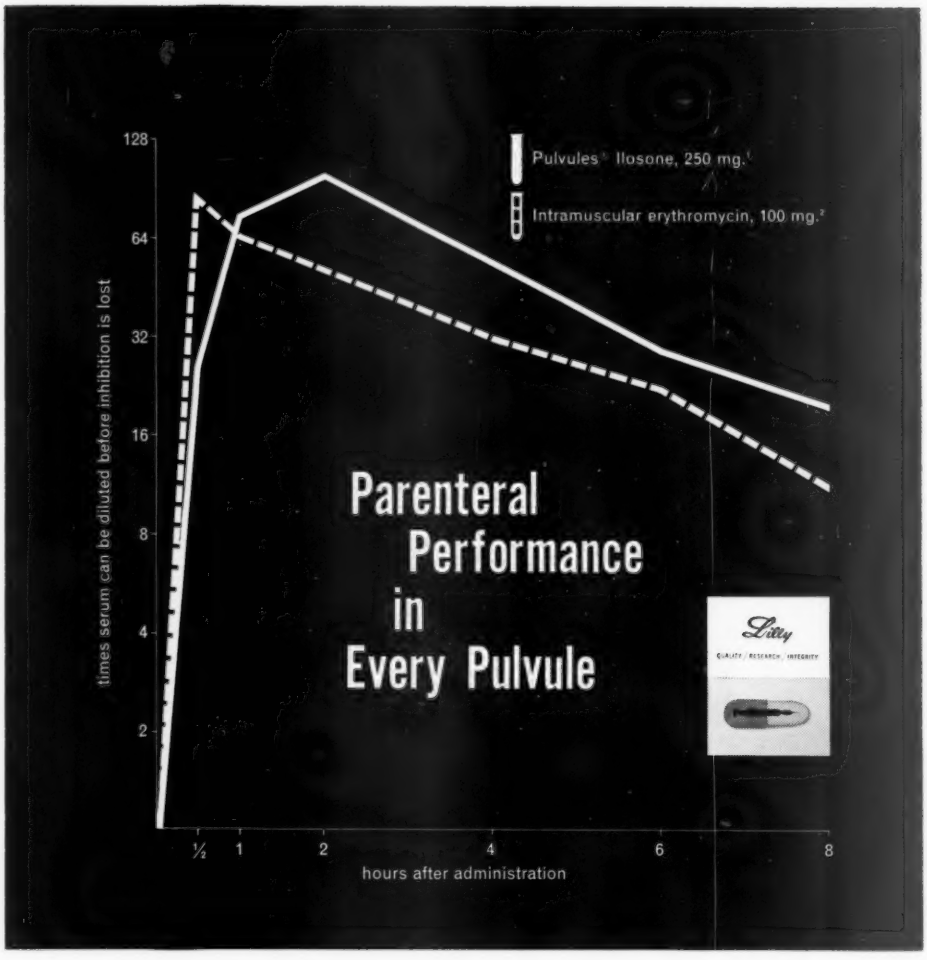

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Parenteral certainty—In more than a thousand determinations, in hundreds of patients studied, Ilosone has never failed to provide significant antibacterial levels in the serum.

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1. Antibiotic Med. & Clin. Therapy, *5*:609, 1958.
2. Data from Antibiotics Annual, p. 269, 1954-1955.

Ilosone™ (propionyl erythromycin ester, Lilly)

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Journal of the
AMERICAN MEDICAL WOMEN'S ASSOCIATION

Volume 14 MARCH, 1959 Number 3

EDITORIAL

Should Wounds Be Covered?

SINCE TIME IMMEMORIAL patients have wrapped their traumatic wounds with some sort of occlusive dressing and physicians have covered all types of surgical wounds. It seemed logical to protect a wounded surface. It was believed that the dressing would keep dirt and bacteria away from the damaged area and thereby hasten healing. Also, the covering had a good psychological effect on the patient.

Recently, however, our convictions on applying dressings to surgical wounds have been shaken. At a recent meeting of the American College of Surgeons, Dr. Ian G. MacDonald and other surgeons of the University of Southern California reported on the advantages of the exposure-suction method in 440 cases of surgical wounds. They reported healing by primary union in 85 per cent of these cases within 21 days. This was especially gratifying in regard to large wounds following radical mastectomy and groin dissections, which previously took one or two months for complete healing. The technique was simple exposure of these large surgical wounds to the air and sunlight in a clean environment. In addition, suction of any serosanguineous fluid was accomplished by a catheter in the operative site. The catheter is hooked up to a negative pressure suction pump. The average small, clean surgical wound requires no dressing or suction.

When we consider that an occlusive dressing provides warmth, decreases oxygen supply, collects serum, and acts as a moist poultice for bacterial growth, we soon realize that wound dressings may be evil indeed. In contrast, exposed wounds have a lower temperature (2 degrees) and are dryer, cleaner, and more comfortable to the patient. All factors in exposed wounds are less favorable to bacterial growth and evidence is rapidly accumulating that surgical wounds heal faster when exposed. This increase in healing rate is probably the chief advantage of the exposure method, but, in addition, raw edges of an exposed wound seal over in several hours while covered wounds seem to take days.

While it must be admitted that open wounds have an adverse psychological effect on some sensitive patients, the advantages of the exposure therapy certainly outweigh this disadvantage. Patients can be educated to this new thinking and in most instances will cooperate with their physician.

It is interesting to note that in Russia the exposure treatment of wounds has been practiced for over a decade. Probably it began with a shortage of dressing supplies, for Russian hospitals function with a minimum of dressings, pads, and bandages. American surgeons are rapidly accepting this new technique and it seems likely that the practice will spread.

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Further Observations on a Living "Decorticate" (Hydranencephalic) Child

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In a previous communication by Kuhlenbeck and others [1] in 1957, general comments as well as anatomic and clinical observations on 3 cases of hydranencephaly were presented. One of these cases, concerning a living child of about 2 years at the time of writing that report (September, 1956), was of particular interest because the diagnosis had been established approximately seven weeks after birth and because repeated succeeding examinations of this child were undertaken.[2] Under conditions of unusually good maternal care, this "decerebrate," or rather "decorticate," but otherwise physically thriving child has reached the age of 3 years and 10 months (at the time of the last examination in July, 1958).

In view of the very substantial brain malformation, closely approaching the severity of the cerebral agenesis in Edinger and Fischer's [3] case (1913) and in Gamper's [4] case (1926), the duration of life in our case appears of interest. The infant in Gamper's case (arhinencephaly with encephalocele) lived about 3 months, and the child in Edinger and Fischer's case (complete lack of differentiated cerebral hemispheres) died before completing 4 years. Supplementing the previous report, we therefore would like to present some additional observations and comments on our case of hydranencephaly.

Discussion and Review of Literature

The diagnostic term hydranencephaly refers to an essentially congenital condition characterized by a highly deficient development or status of the cerebral hemispheres, compatible with a fairly normally developed skull. The intracranial space left by the telencephalic defect is filled by cerebrospinal fluid, which may be intraventricular or extraventricular, or both. The clinical diagnosis of hydranencephaly, although justified and at times the only possible diagnosis, is not particularly satisfactory, since it rests on a merely incidental feature and does not refer to the different etiological factors or to the different anatomic manifestations related to hydranencephaly as the common feature.

It has been justly stressed that in analyzing the origin of brain malformations, causal origin or pathogenesis must be distinguished from formal origin or morphogenesis.[5] In other words, the relevant functional variables and the relevant formative processes as well as changes must be separately considered. In Ashby's [6] terminology the former represent the operators, the latter represent the set of morphologic transitions, on a set of operands, namely structures of the neuraxis, resulting in the transformation.

Combining these viewpoints it may be said that hydranencephaly can be classified into two main types: (1) that caused by intrinsically arrested or defective developmental morphogenetic and histogenetic processes, and (2) that caused by extrinsic destructive encephaloclastic processes, undoing the results of normal histogenesis. Since the processes subsumed under both forms might set in at any stage of morphogenesis, and since the processes under form 1 and 2 are not definable in fully rigorous fashion, intermediate forms of 1 and 2 might perhaps occur.

An unequivocal and typical instance of form 1 is the case of arhinencephaly described by Kuhlenbeck and Globus in 1936 [7]; typical instances of form 2 are the 2 cases carefully studied and evaluated by Lange-Cosack in 1944.[8] The case reported by Jakob in 1931 [9]

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likewise seems to belong to that form. In regard to a number of other cases recorded in the literature, especially some of the earlier ones, it is not always possible, on the basis of the data and illustrations published by the authors concerned, to reach a definite conclusion, since many of the relevant morphogenetic and neuropathological concepts were not in all instances uniformly defined or sufficiently clarified.

In cases of form 2 hemosiderin may be found diffusely in meninges, the choroid plexus, and the glial margin, justifying the inference of a destructive process involving hemorrhages. Lange-Cosack [8] believes that traumatic damage or various circulatory disturbances play an important role. This latter possibility is corroborated by the experiments of Becker,[10] who produced vesicular malformations of the prosencephalon ("Blasenhirn") closely resembling certain forms of hydranencephaly and porencephaly by blocking branches of the internal carotid in young dogs.

In cases of form 1, the brain malformation may display features of arrested and incomplete development, maintaining, however, essentially normal and orderly structural and topologic relationships. In other cases of this form gross distortion and randomness of structural arrangements obtain, displaying a nearly teratomatous type of growth. Nothing certain can be inferred concerning causal factors. It is, however, of interest that Giroud and others [11] in 1957, in subjecting female rats to a diet deficient in pantothenic acid before and after mating, observed malformations of the brain in the embryos. In some of these malformations, the telencephalon was not subdivided into two separate hemispheres and it displayed features resembling cyclencephaly as it occurs in man. In contradistinction to some of these human malformations, cranial development was also disturbed. Although nutritional factors may thus represent a significant variable in such malformations, there is little probability that a deficiency of that type played a role in our case as well as in most other similar cases. Radiational damage might also be invoked, but again without any relevant probability.

In our case and in other similar cases the disturbance must have manifested itself at a stage in which continued normal telencephalic development does not represent an essential factor upon which further normal formative processes of meningeal and cranial development, as well as cranial growth, depend. In 1957 Bettinger,[12] who had previously (1940) [13] recorded and discussed observations on hydranencephaly, commented on a puzzling aspect of this condition: On the one hand, the normal size of the head and the perfect development of the cranial vault makes one inclined to put the date for the commencement of the disturbance as late as possible in pregnancy, probably considerably later than the fifth month; on the other hand, the later the date, the more difficult it becomes to account for the complete dissolution of a fair amount of the brain substance, which must have been present. To this latter remark of Bettinger's, which would apply particularly to cases of form 2, it should be added that, the later the date, the more difficult it becomes to account for a lack of corpus callosum and for the manifestation of a univentricular telencephalon in the cases of form 1, in which secondary dissolution of brain substance cannot, as a rule, be assumed. It becomes necessary then to infer that some phases of the disturbance would have manifested themselves already during the sixth or seventh week of embryonic development. Yet, such telencephalic growth disturbances must have been compatible with normal cranial and meningeal development. This, again, would imply that, at least in man, telencephalic and cranial development might, under certain circumstances, proceed independently of each other with a surprisingly early dichotomy.

In 1 of the 2 other cases cited in the previous communication,[1] in which development was clearly arrested and incomplete, some fresh hemorrhages, without any evidence of old ones, were found in the telencephalon. These hemorrhages could not possibly have caused the malformation and were presumably results of birth trauma. Although Lange-Cosack [8] justly stresses the possible causal significance of hemorrhagic processes, due caution as regards this interpretation, and particularly the evaluation of moderate amounts of hemosiderin in older cases, seems advisable.

Additional remarks on various aspects of cyclencephalic and related cerebral malformations can be found in Ostertag's [14] comprehensive survey of this general subject.

It has been justly emphasized that at birth an infant gives no conclusive evidence of functioning cerebral hemispheres or, better, of cortical mechanisms and that the newborn infant

can thus, to a certain extent, be likened to a reflex animal experimentally deprived of hemispheres.[15] If no conspicuous anomaly of the head is noticeable, malformations of the telencephalon related to hydranencephaly usually are not detected at birth or during the first weeks of life. Such infants behave "absolutely in the same way as other babies during the first week of life."[13] Or, as others have remarked, it is unlikely that cortical defects may be recognized before cortical function can exert its influence on behavior. After a variable number of weeks failure to develop the normal postural, locomotor, and general behavioral patterns then becomes clearly noticeable.

Before discussing our subsequent observations on the "decorticate" child, the following brief summary of our case is given.

SUMMARY OF CASE

J. M., a Negro girl, was born by spontaneous delivery on Sept. 27, 1954. No abnormality was detected upon her discharge examination. Two normal siblings, a girl now aged 14 and a boy now aged 11, are living.* Both are very bright children, and the boy has an outstanding school record. The medical history of the parents did not include any relevant data concerning hereditary or other pathological factors.

The child appeared to develop normally; however, upon examination during a routine clinic visit on Nov. 1, 1954, her head seemed slightly smaller in relation to her body than might be expected, although it was still within normal limits. On the next visit, Nov. 18, 1954, a moderate umbilical hernia was apparent. The neurological examination now evidenced definite symptoms of decerebrate rigidity. The child was lying in opisthotonus with rigidly extended limbs. Reflexes were difficult to elicit because of the marked rigidity, but, if manifested, appeared exaggerated. Tonic grasp and neck reflexes were present. To most stimuli the typical response was rigid opisthotonus, with limbs extended and hands clenched. The upper extremity was extended, adducted, and internally rotated at the shoulder joint. The forearm was extended at the elbow and hyperpronated (Fig. 1*A*). The wrist was usually extended in the metacarpophalangeal joints and flexed at the interphalangeal joints. The lower extremity was adducted and extended at the hip, knee, and ankle, and the toes were in plantar flexion. Together with opisthotonus, and especially when the child was supported in the prone position across the lower chest and abdomen, this pattern of rigidity gave a typical picture appropriately designated as "swan dive" (Fig. 1*B*). Although this pattern could be elicited without holding the head of the child in strong dorsiflexion, such dorsiflexion, as shown in the illustration, would markedly facilitate, increase, and prolong the rigidity.†

The ophthalmoscopic picture was normal, and the pupillary light reflex was present. However, the child did not react to other visual stimuli; the eyes moved at random in parallel conjugation and never manifested convergence. It was also doubtful whether the child could hear.

A pneumoencephalogram by spinal puncture disclosed a grossly malformed, aplastic cerebrum. The entire neopallium seemed to be reduced to a flabby bag presumably containing a single ventricular space. The relevant roentgenograms were shown in the first publication.[1] The essentially similar results of a new pneumoencephalographic exploration performed in May, 1958, about three and a half years later, will be discussed later in this communication.

Diagnostic Comment. While there could not be any doubt about the diagnosis of hydranencephaly, it should be emphasized that, without actual gross and microscopic anatomic corroboration, our specific diagnosis (univentricular telencephalic malformation) can only be tentative, although rather probable. This probability rests on the fact that it is possible

*The inaccurate age figures of (then) 13 and 11 years given in the previous report (1957) resulted from a trivial lapsus of computation.

†Fig. 1*A* and 1*B* are "stills" from a motion picture prepared and edited in joint co-operation by the departments of pediatrics, medicine, radiology, physiology, and anatomy of the Woman's Medical College of Pennsylvania.

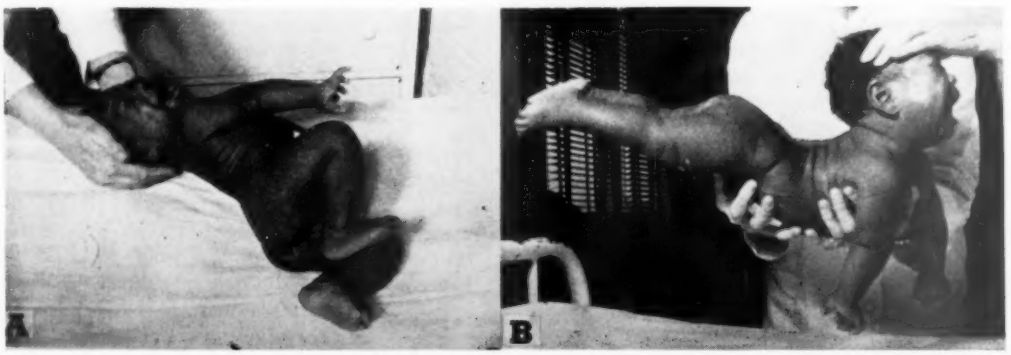

Fig. 1 (*A*). Arm position of child upon homolateral passive rotation of head. Note unusually strong flexion of right lower extremity. No spontaneous Babinski (November, 1954). (*B.*) Swan-dive position of lower extremities. Note arm positions and clenched hands (November, 1954).

to interpret the pneumoencephalographic picture in full conformity with the anatomic features studied and described by Kuhlenbeck and Globus [7] in a case of arhinencephaly. An alternate, although presumably less probable, possibility would be a prenatal encephaloclastic process, essentially restricted to the prosencephalon, resulting in softening, resorption, and shrinkage, and corresponding to the cases studied by Lange-Cosack [8] or somewhat related to the similar condition in Jakob's case. [9]

In regard to the subsequent history of the case, the child was readmitted in April, 1955, because of a temperature of 103 F. Examination did not reveal a cause for this fever. The neurological condition was unchanged, and the umbilical hernia had increased somewhat. Weight, height, and head proportions were within normal limits. She was hospitalized for a week and showed temperature spikes of 101 to 103 F on all but two days. It was concluded that the fever was of cerebral origin, and she was then discharged.

The child was again seen and examined in June and September, 1956. At both times she appeared well developed and well nourished. The umbilical hernia had remained stationary. Alternating periods of rest (sleep) and of activity (wakefulness) occurred in a fairly regular, alternating rhythm. Occasional periods of apparently moderate "fever" were reported by the mother. The child was unable to right herself or to stand with moderate support; she was unable even to hold her head upright in a normally extended position. The head flopped helplessly forward, backward, or sideward by its own gravity.

The child had no adequate behavioral reaction to her surroundings, so that it could be inferred that she did not "recognize" persons or places. Although the pupillary reaction to light remained normal, she did not fix her gaze or follow objects with the eyes. It thus was assumed that she did not "see" in the generally accepted sense of the word. She did show certain motor reactions to sounds, but, here again, it appeared that she did not "recognize" sounds. It could not be conclusively ascertained whether she had an olfactory sense, but it appeared probable that she did not.

Muscular rigidity still obtained, but the pattern at that time had changed from the type of manifest decerebrate rigidity to that of spastic paraplegia with marked spasticity of the adductors.

The child was still irritable to stimuli, although much less than before, and cried easily upon manipulation. She was unable to utter any other sounds than crying sounds, accompanied by corresponding facial expressions. She was most easily quieted by being held in a position of flexion of the trunk and lower extremities, with slight flexion of the head, against the examiner's chest. If the proper position were maintained, it was irrelevant whether the mother or a stranger held her in that way. Speaking to the child in a monotonous soothing voice had a marked contributory quieting effect during this procedure, regardless of whether it was the voice of the mother or of a stranger.

Unfortunately, because of the more comprehensive pneumoencephalographic examinations, the simpler transillumination test of the head had been omitted in November, 1954. It was now performed, with negative results, but it is, of course, not possible to state whether it would have been positive in 1954.

On the basis of the original roentgenographic picture of November, 1954, which again is corroborated by the recent roentgenray findings of May, 1958, to be discussed and depicted later in this paper, the cerebral defect involves mainly the pallium, that is, the cortex telencephali. Diencephalon and basal ganglia, although doubtless functionally and presumably also morphologically (structurally) affected because of the lack of normal cortical connections and control, appear anatomically to be much less implicated in the obtaining cerebral aplasia. From the anatomic viewpoint, the child is decorticate rather than decerebrate. The results of electroencephalography, likewise to be discussed and depicted later in this paper, can be interpreted in agreement with this view. In the previous communication the problem of consciousness was briefly referred to; it was stated that the child might not experience any consciousness or awareness at all. This opinion still can be upheld at the present time, although, as had already been emphasized in 1957, such opinion is merely a conjecture, since consciousness of an observed living organism remains for the observer an inference from behavior and is not directly observable. In the terminology of one of us (H.K.),[16] consciousness is synonymous with mind *sensu latiori*, while the term "mind *sensu strictiori*" applies to particular aspects of consciousness such as manifested by emotions and thoughts. For some such manifestations, the term "higher symbolizing consciousness" was suggested, and this latter could also be designated as mind *sensu strictissimo*. The inference from behavior that the child is "mindless" in this strictest sense can thus be regarded as most cogent, while the inference that the child is "mindless" in an over-all sense is less cogent but still rather probable.

Although we have in this instance, for all practical purposes, a mindless human being, not essentially differing from a complex mechanical doll, the mother has developed a strong emotional attachment to this child. The mother is a rational and intelligent person who fully understands and realizes the essential features of the situation. Yet, she is convinced that she can, in a fashion, communicate with the child, and that, although in an exceedingly dim manner, the child recognizes her. On the basis of our observations, we are inclined to doubt this and to interpret this attitude of the

mother—of course fully understandable—as a hyponoic and hypobulic delusion.

SUBSEQUENT CLINICAL OBSERVATIONS

Subsequent to the previous report the child was examined in December, 1957, and May, 1958. In July, 1958, she weighed 26 lb., which is a little below normal for a child of more than 3.5 years, and measured 35 in. in "height" (meaning total length). It must be taken into consideration that, since normal use is eliminated, the inferior extremities display a measure of atrophy. Considering, though, the development of head, trunk, shoulders, and upper extremities, the definite impression of a well-nourished child is obtained.

A pneumoencephalographic study was undertaken in May, 1958, and electroencephalograms were obtained in July, 1958. A cerebral angiographic study was suggested, but, in view of its risk and of the afore-mentioned emotional situation, it was rejected; after the pneumoencephalographic procedure the child suffered a severe, shocklike condition and had to remain hospitalized in a serious condition for four days. The mother, upon her request, was allowed to stay overnight in the hospital with her child.

In December, 1957, the child was demonstrated for a seminar in pediatrics. The umbilical hernia had decreased in size so that it was at times unnoticeable upon superficial inspection. The general muscular status was very similar to that of September, 1956. Despite considerable rigidity, particularly involving adductor spasm with crossed extended inferior extremities, the general pattern was more that of spastic paraplegia than that of decerebrate rigidity. Upon stimulation of the plantar surface, a strong Babinski sign could be elicited on both sides. The swan-dive position could be obtained, but with greater difficulty than previously.

Neck reflexes, that is, the statokinetic reflexes from head to body by passive movements of the head, could be elicited and will be more fully described. No reflexes from body to head were noticeable. As before, the child was not able to stand, sit, or raise her trunk. The head could not be righted and simply followed the pull of gravity. It is thus of particular interest that the child, although anatomically not deprived of thalamus and basal ganglia, was unable to manifest the righting reflexes upon neck and head characteristic of a "thalamus animal." She was in this respect, although anatomically "decorticate," functionally also decerebrate.

The pupillary light reflex remained bilaterally normal. This implies an at least partially functioning pretectal region of the thalamus and, most likely, the presence of a lateral geniculate nucleus. While this latter structure is not believed to be directly involved in the pupillary light reflex, this nucleus is intimately related to the optic tract and must be presumed present if a functionally adequate part of the optic tract reaches the pretectal region and superior colliculus. A fairly well-developed lateral geniculate body * was found and illustrated in the case of Kuhlenbeck and Globus.[7] In our present case the randomly parallel conjugate movements of the eye balls, taking place without any convergence, give a somewhat weird effect reminiscent of an automatic mechanism.

In May, 1958, the child was again demonstrated in the regular physiology course for medical students. Although the general physical condition was essentially in accordance with the findings at the previous examinations, the child seemed to be in a condition of more pronounced rigidity, which again could be evaluated as of almost true decerebrate type. Because of the rigidity it was nearly impossible to elicit such reflexes as knee jerk and ankle jerk.

In a supine position, the child would lie with her head dorsiflexed, and, less rigidly extended in strong adduction, the lower extremity crossed scissorlike in the midline (Fig. 2*A* and *B*). The arms were extended or flexed contingent upon the head position, which was purely passive and depended on external manipulation or gravity. As before, there was complete inability to assume a normal head position. The righting reflexes (visual and static labyrinthine) were completely lacking.

The Magnus-de Kleijn reflexes of upper extremities upon passive rotation of the head were easily demonstrated. The typical response by ipsilateral extension and contralateral flexion is shown in figure 2*C*. With

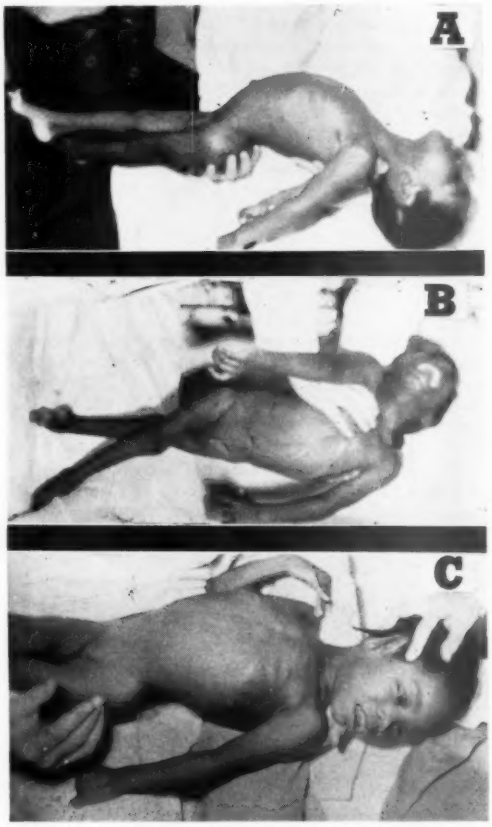

Fig. 2 (*A* and *B*). Manifestations of rigidity with child in supine position (May, 1958). The spontaneous Babinski is particularly noticeable in *A*. (*C*). Demonstration of Magnus-de Kleijn reflex (May, 1958).

*It is of particular interest that this lateral geniculate body (Fig. 16)[7] was rotated 180 degrees, that is, completely reversed, around its rostro-occipital axis.

the child's head held in dorsiflexion, the extensor rigidity was markedly increased, and by holding the child in the appropriate position a typical swan-dive pattern was obtained easily. Both the "lengthening and shortening reaction" of the extensor muscles could be easily demonstrated. It is of particular interest that the toes of both feet were in a continuous Babinski position with conspicuous fanning. The Babinski sign, which could be demonstrated freely in December, thus could not be truly elicited as a response now, because the position was continuous.

In regard to the pupillary light reflex and the conjugate but not converging eye movements, the findings were identical with those of the previous examinations. Concerning the complete lack of any behavioral reaction attributable to a recognition of the environment, there was likewise no change.

The alternating periods of sleep (rest) and wakefulness (activity) at the time of the last examinations (May and July, 1958) manifested approximately the same normal rhythm described in the first communication. The child is maintained under mild sedation with phenobarbital. She is still spoon-fed with baby food and does not masticate but uses exaggerated sucking movements; a full set of deciduous teeth is now present.

Urination occurs automatically at irregular intervals in response to pressure. Emptying of the bowels is obtained by enemas. The mother claims that regular or "normal" defecation cannot be performed. In this respect it is perhaps of interest to recall that in another of the 3 cases of hydranencephaly (case 2) reported in the preceding communication,[1] concerning a boy aged 3 years, one of the prominent symptoms was said to have been "intractable obstipation."

RESULTS OF PNEUMOENCEPHALOGRAPHY

The pneumoencephalogram by spinal puncture, performed in May, 1958, revealed findings essentially similar to those obtained in November, 1954. Large amounts of air filled the cranial cavity, indicating a malformed, aplastic cerebrum (Fig. 3-8). In the region of the telencephalon, a large air bubble was present, which changed its location according to the position of the head. Figure 3 shows the complete collapse of the occipital lobe with the head in a prone position, whereby the entire flabby telencephalon sagged rostralward; on the other hand, in this position, presumably because of subtentorial counterpressure, the cerebellum remained rather firmly in place. In the series of pneumoencephalograms done in 1954 it was not possible to ascertain whether the air bubble was in the telencephalic ventricle or in the subarachnoid and subdural space. In the present series, figure 4 shows conclusively that on the right side the air is within the ventricular space and on the left side it is within both the ventricular space and the subarachnoid (and perhaps subdural) space. Since the obtained roentgenographic outlines of the

Fig. 3. Pneumoencephalogram, left lateral view, with child's head in recumbent (prone) position. Note unerupted permanent first lower molar, also visible in some other views (for example, the right, left, upper, and lower in figure 6*A*). The numbers and letters signify the following: 1=cerebellum, 2=subtentorial space, 3=cisterna ambiens, 4=cisterna chiasmatis, 5=cisterna interpeduncularis, 9=mastoid cells, 10=cisterna olfactoria, l=subluxations of atlas and axis, and t=tentorium.

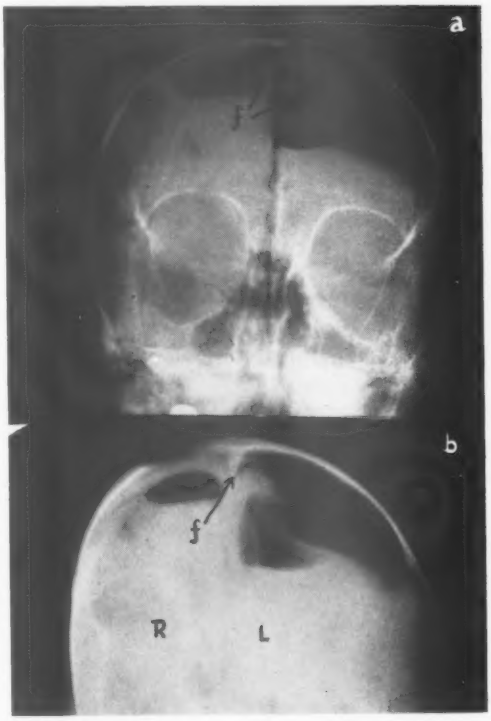

Fig. 4. Pneumoencephalogram, anteroposterior view, with child's head in erect position. *B* is underexposed, showing, by greater contrast, more details of flabby, membranelike pallium, which has partly collapsed on left side (right side of figure). The letters signify the following: f=rudiment of falx cerebri, R=right, and L=left.

Fig. 5. Pneumoencephalogram, left lateral view, with child's head in semierect position. Note optic nerve in cisterna chiasmatis. Some of the dark lines in cerebellar region represent diploic venous channels. The numbers and letters signify the following: 1=cerebellum, 2=subtentorial space, 3=cisterna ambiens, 4=cisterna chiasmatis, 5=cisterna interpeduncularis, 7=optic nerve, 8=cisterna pontis, 9=mastoid cells, l=subluxations of atlas and axis, and t=tentorium.

pneumoencephalograms of 1954 and 1958 can easily be interpreted as conforming to the brain malformation depicted by Kuhlenbeck and Globus in 1936,[7] the presence of two separate air bubbles in the frontodorsal recesses does not preclude the likelihood of a univentricular (cyclencephalic) telencephalic malformation. The air bubbles shown in figures 5 and 6 (lateral views) presumably correspond closely in position to the air bubbles shown in figure 4 (anteroposterior view). It is probable that the air entered the ventricular space through the large membranous roof of the horseshoe-shaped brain, as depicted in the previous communication [1] and in serial outline drawings.[7] With the head in the prone position (Fig. 3), the air escaped again through this membranous roof and was entirely extraventricular, with collapse of the occipital lobe. With the head in the supine position, the anteroposterior view (Fig. 7) again shows air in one frontal ventricular recess, and air in the frontal ventricular recess as well as in the subarachnoid (and perhaps subdural) space on the other side.

Interesting outlines are shown in figure 8*A*. The large ovoid radio-opaque mass presumably represents the basal ganglia (particularly the caudate nucleus) superimposed upon the diencephalon. Above it, air presumably is contained within a common telencephalic ventricle, lined by a curved row of regular bubbles. These bubbles may well be caught between the tufts of a choroid plexus arranged in conformity with that of the outline drawings of Kuhlenbeck and Globus.[7] In the frontal region, in which a choroid plexus is not present, the air bubbles assume a changed pattern. Fig-

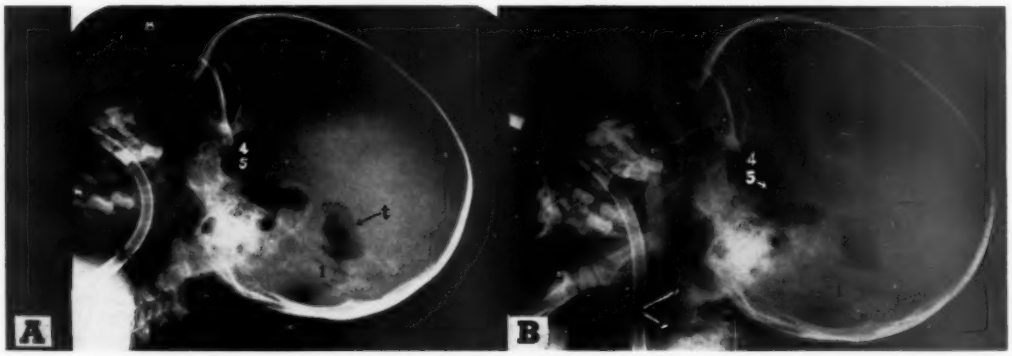

Fig. 6. Pneumoencephalograms, with child's head in semirecumbent (supine) position. (*A*). Right lateral view. (*B*). Left lateral view. The numbers and letters signify the following: 1=cerebellum, 2=subtentorial space, 3=cisterna ambiens, 4=cisterna chiasmatis, 5=cisterna interpeduncularis, 8=cisterna pontis, 9=mastoid cells, 11=air in region of vallecula cerebelli, l=subluxations of atlas and axis, and t=tentorium.

Fig. 7. Pneumoencephalogram, anteroposterior view, with child's head in recumbent position (left side of head on right side of figure). The numbers and letters signify the following: 2=subtentorial space, 6=air in rudimentary longitudinal cerebral fissure between frontal portions of cyclencephalic brain, 11=air in region of vallecula cerebelli, L=left, and R=right.

ure 8*B* displays a different distribution of the air, which was presumably entirely extraventricular.

The basal cisterns are, as before, greatly enlarged the cisterna chiasmatis, interpeduncularis, pontis, and ambiens merging into each other. The optic nerve can be seen passing through the cisterna chiasmatis in figures 5 and 8. Much air is likewise present in the cisterna magna and subtentorial space, but the cerebellum, although small, is not significantly below the lower limits of normal.

In addition to the pneumoencephalographic findings, the roentgenographic pictures revealed a condition not detected by the previous examinations, namely, a very pronounced, mostly anterior subluxation of the atlas against the axis (Fig. 5, 6, and 8*A*) and, to a lesser degree, a posterior and anterior subluxation of the axis against the third cervical vertebra (Fig. 6). It appears almost incredible that the spinal cord does not clinically manifest any damage from the obvious displacement affecting the cranial part of the vertebral canal. These subluxations are evidently related to the fact that no righting reflexes of the head occur and that, therefore, no muscular control of the flopping head can be accomplished. The uncontrolled movements of the head as pulled by its own weight presumably greatly loosened the articular connections between atlas, axis, and third vertebra but apparently did not affect those between occipital bone and atlas.

ELECTROENCEPHALOGRAPHIC RECORDING

In July, 1958, an electroencephalographic

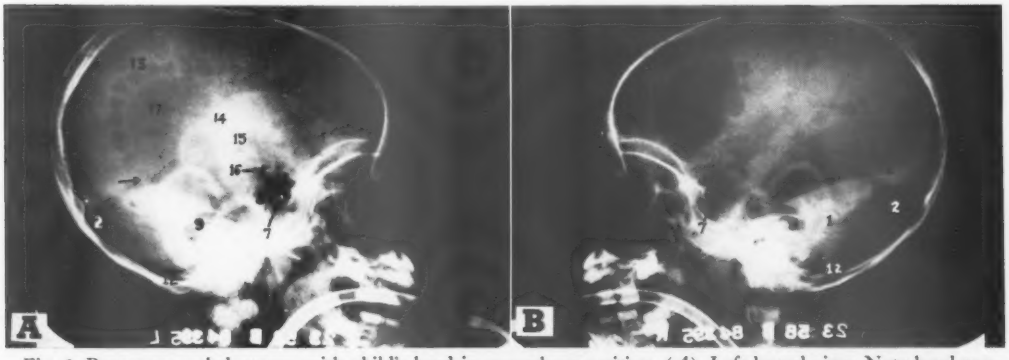

Fig. 8. Pneumoencephalograms, with child's head in recumbent position. (*A*). Left lateral view. Note basal ganglia and diencephalon, optic nerve, and bubbles along choroid plexus (?). (*B*). Right lateral view. Note optic nerve. The numbers and letters signify the following: 1=cerebellum, 2=subtentorial space, 3=cisterna ambiens, 4=cisterna chiasmatis, 5=cisterna interpeduncularis, 7=optic nerve, 8=cisterna pontis, 9=mastoid cells, 12=cisterna magna, 13=air bubbles between tufts of choroid plexus (between arrows), 14=basal ganglia (caudate nucleus), 15=thalamus, 16=hypothalamus and part of third ventricle, 17=air in common telencephalic ventricle, l=subluxations of atlas and axis, and t=tentorium.

examination was performed.* The child was held with the head in an upright position, with flexion of trunk and lower extremities, against the mother's chest and in the mother's arms. Electrodes were placed over the frontal, "motor," parietal, anterior temporal, middle temporal, and occipital regions bilaterally; monopolar and bipolar recordings were taken.

A fairly regular slow activity, with a moderate synchronization between right and left, was registered. In some parts of the record the frequency in the occipital region was higher than in the frontal region. The predominating rhythm in the frontal region was 3 to 4 cps, with voltage up to ± 200 μv in each direction, while the rhythm in the occipital region was

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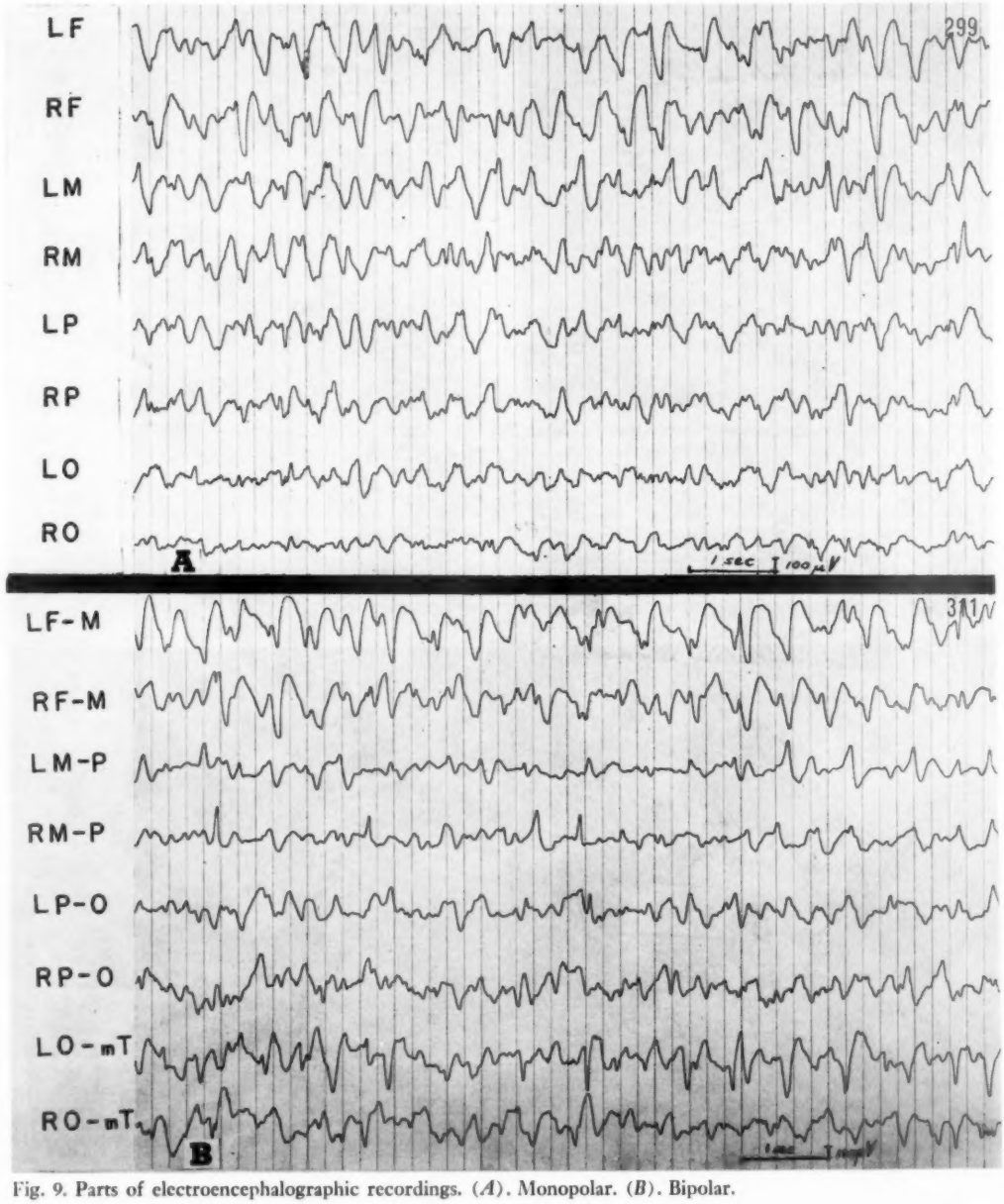

Fig. 9. Parts of electroencephalographic recordings. (*A*). Monopolar. (*B*). Bipolar.

predominantly 4 to 5 cps, with a lesser voltage of up to about ± 100 μv in each direction. In the occipital region there was a tendency toward superposition of occasional runs of faster frequencies up to more than 10 cps; these smaller waves lasted less than half or one third of a second and had a lower voltage of approximately 50 μv or less in each direction. Occasionally, isolated, fairly high-voltage sharp waves or spikes (± 200 μv), at times clearly shown especially in the bipolar recordings, would occur in all regions. Figure 9*A* shows part of the monopolar recording, and figure 9*B* illustrates part of the bipolar one.

In view of the severe cortical malformation, anatomically demonstrable by the pneumoencephalogram, and, moreover, corroborated by the clinical picture, including the apparently complete cortical blindness, it can be inferred that the conspicuous rhythmic and, in part, highly synchronized recording represents essentially the electrical activity of basal ganglia and diencephalon, transmitted through the parenchyma of the flabby sacklike pallium and the cerebrospinal fluid.

In the first case of the preceding communication,[1] in which a detailed histological examination had been made,[7] a rudimentary neocortex of fetal type, corresponding in some respects more or less to the sixth fetal month, was found. This cortex showed in somewhat irregular fashion the six-layered pattern, with the fourth layer incompletely distinguishable from the inner part of the third layer. This undifferentiated cortex consisted mostly of immature cells resembling neuroblasts. Although it can be inferred most cogently that such a cortex could not perform significant neural functions to any appreciable degree, the possibility must be considered that some neural connections had been established and that some electrical activity might have occurred. In addition, it must be stressed that rhythmic electrical discharges can be manifested by a cortex completely isolated from all afferent and efferent connections.[17] Assuming that in our present case a structural arrangement somewhat similar to that of the cited previous case obtains, some actual participation of the cortical rudiments in the observed electrical activity cannot be entirely excluded. Thus, for instance, the occurrence of high-voltage discharges is known to be a consequence of isolation of the cortex.[18]

SUMMARY

Recent clinical, pneumoencephalographic, and electroencephalographic findings in a living "decorticate" hydranencephalic child of about 4 years of age, whose condition was diagnosed seven weeks after birth, are discussed, and a previously published report concerning the findings in the child at slightly over 2 years of age is summarized.

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Profound Hypoglycemia

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PROFOUND HYPOGLYCEMIA is an uncommon occurrence during insulin therapy for diabetes mellitus; it is seen after a patient takes insulin with a suicidal intent; it may occur in the course of insulin shock therapy; it is occasionally the presenting clinical picture in anterior pituitary insufficiency; it may be a manifestation of insulinoma; and it has been seen after ingestion of an oral hypoglycemic agent.[1] This list does not exhaust the clinical situations in which hypoglycemia may be present and significant, but it emphasizes those situations in which the degree and duration of hypoglycemia are most commonly fatal. The term "irreversible hypoglycemia" has been applied to those cases with a lethal outcome—in spite of the fact that ante-mortem blood glucose levels had often been restored to normal or above—because post-mortem examination has failed to reveal any other cause of death.

The physiological range of blood glucose for all ages and for all times of a 24 hour period is 40 to 150 mg. per 100 cc. (Folin-Wu) or 20 to 130 mg. per 100 cc. of true blood glucose. The extreme low of 20 mg. per 100 cc. is encountered in the neonatal period [2,3]; a level of 40 mg. per 100 cc. (Folin-Wu) may be seen in asymptomatic diabetic children,[3] but it is unusual to find a blood glucose level below 60 mg. per 100 cc. (Folin-Wu) in adults.[4] Among nondiabetic individuals symptoms may be associated with levels below 60 mg. per 100 cc. (Folin-Wu) and are almost always present with levels below 50 mg. per 100 cc. Insulin-takin diabetic patients often have symptoms at levels well above these figures, however; this is in part because they are accustomed to higher levels and in part because a rapid rate of fall of blood glucose (as after insulin administration) may provoke symptoms without having induced absolute hypoglycemia.

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The mechanism of death in profound hypoglycemia appears to be irreversible cerebral damage. In both profound hypoglycemia and diabetic ketosis, the cerebral respiratory rate is depressed. At 20 mg. per 100 cc. blood glucose the rate is 24 per cent below normal; at 9 mg. per 100 cc. it is 44 per cent below normal.[5] Fatal hypoglycemia has been found to be associated with an even greater lowering of cerebral respiration; at this point neither glucose nor oxygen will restore it.[5] Utilization of oxygen by tissue is dependent upon respiratory catalysts whose integrity is conditioned by the glycolytic cycle; the oxygen deficit in this instance is secondary to the glucose deficit. In the final analysis there is no alternative substrate to glucose for respiration of brain tissue.

Cerebral edema, petechial hemorrhages, and cellular damage with cell degeneration have been found in post-mortem studies of fatal hypoglycemia in the human.[3, 6] The degree of individual sensitivity to hypoglycemia probably depends upon variation in details of biochemical organization such as occur with variation in age, nutrition, disease processes, and so forth, insofar as these are reflected in cerebral metabolism. From a clinical point of view any blood glucose level below 50 mg. per 100 cc. (Folin-Wu) should be considered an emergency and one below 40 mg. per 100 cc. potentially critical. It should be recalled in this connection that the Folin-Wu method for determining blood glucose includes 19 to 31 mg. per 100 cc. non-glucose-reducing substances,[7] so that a Folin-Wu reading of 40 mg. per 100 cc. might mean a level actually as low as 9 mg. per 100 cc.

The treatment, of course, is glucose administration, and the essence of successful therapy is enough available glucose soon enough. The avidity of tissues for glucose during profound hypoglycemia is astounding. The minimum rate of glucose administration should be 150 Gm. of glucose per hour; the total dose of glucose required may be as great as 5 Gm. of glucose per unit of insulin received. One hundred and fifty grams of glucose per hour, assuming

a blood volume of 5 liters, endogenous glucose of zero, and tissue avidity for glucose such that all available blood glucose is immediately taken up by the tissues, would yield a blood glucose level of 50 mg. per 100 cc. per minute if there were no glucosuria; if glucosuria should amount to as much as one third of administered glucose, as it may with very rapid rates of diuresis, the blood level would be 34 mg. per 100 cc. per minute. Thus, under the most adverse conditions, a rate of glucose administration of 150 Gm. per hour could be expected to maintain a blood glucose level just compatible with full protection of the central nervous system from that point on. In practice, when the situation is extreme, this rate of glucose administration does not induce marked hyperglycemia. In the 2 most extreme cases that have occurred since adoption of this regimen, each patient required a total dose of 1,700 Gm. of glucose: one patient had gone into profound hypoglycemia on the third day of receiving 3,000 units of insulin daily during insulin shock therapy, and the other had received an oral hypoglycemic agent. Neither patient was diabetic; both survived their episodes. In terms of the amount of glucose required per unit of insulin received, it seems to be the diabetic patients whose resistance is least. This is not as odd as it seems, in view of the fact that continued administration of insulin overcomes insulin resistance and that the dose of insulin for a diabetic patient is necessarily one to which he will respond; in other words, his resistance, if present, usually has already been broken.

When glucose is administered at a rate of 150 Gm. per hour, the blood glucose level generally does not become excessive until the body has regained its ability to maintain its own blood glucose level. When blood glucose reaches 150 to 200 mg. per 100 cc., the rate of administration may be slowed but the level must not be allowed to lapse back into the hypoglycemic range. Whether or not the patient is responsive at this point, and, if so, the degree of his responsiveness, is highly variable; it is probably chiefly dependent upon the degree and extent of the cerebral changes that occurred during the profoundly hypoglycemic interval. Relapse should be expected; it may not occur but most usually does. The situation in respect to blood glucose cannot be considered corrected until the body has maintained normal blood glucose levels with only the usual daily oral feedings for a 48 hour period.

Restoration of the body's capacity to maintain normal blood glucose levels does not guarantee ultimate recovery. Two patients, 1 male and 1 female, seen recently, both diabetic and in their seventies, had received their usual daily dose of insulin and were admitted to the hospital with blood glucose levels under 30 mg. per 100 cc. (Folin-Wu) and coma of at least several hours' duration. Both were treated intensively and restored to full consciousness and orientation without residual motor impairment; each had spontaneously maintained blood glucose within the normal range for at least 24 hours, only, at that point, to become hypotensive. Hypotension was corrected by continuous norepinephrine administration, but both patients died. Post-mortem examination was made in 1 case and revealed only those changes associated with profound hypoglycemia. This variety of delayed hypotension has not yet been seen by the author in younger patients, although duration of coma prior to therapy in some cases has been comparable.

Utilization of glucose requires the presence of certain members of the B complex group of vitamins. The ascorbic acid of the adrenal cortex is rapidly depleted in conditions of severe stress. There is a question of responsiveness to adrenal steroids during prolonged insulin coma, but also evidence that some individuals who fail to respond to glucose alone will recover if ACTH or cortisone is given and if blood glucose is at least normal.[8] Certain metabolic processes related to carbohydrate metabolism are accompanied by transfer of potassium into cells.[9] Glycogen storage is associated with retention of 0.36 mM. of potassium per Gm. of glycogen.[10] Because these substances play a role in the biochemical usableness of glucose in tissues, they have been included in the regimen for profound hypoglycemia. The exact amounts required are not known; the current regimen is as follows:

1. Give 50 cc. of 50 per cent glucose in distilled water with 5 mEq. of potassium intravenously. Repeat every 30 minutes.

2. Insert airway.

3. Give 1,000 cc. of 10 per cent glucose in distilled water intravenously, to which has been added:

Thiamine hydrochloride, 100 mg.
Riboflavin, 10 mg.
Nicotinamide, 100 mg.
Pyridoxine hydrochloride, 50 mg.

Calcium pantothenate, 10 mg.*
Ascorbic acid, 100 mg.
Cyanocobalamin, 100 μg
Hydrocortisone, 100 mg.
Potassium chloride, 20 mEq.

4. Pass stomach tube and instill 50 cc. of 50 per cent glucose with 5 mEq. of potassium every 30 minutes.

5. Give 100 mg. of hydrocortisone intramuscularly.

6. For temperature of 103 F or above use tepid sponge or alcohol sponge. Use cold packs if necessary.

7. When the patient is able to take food orally and to retain it, give:

One glass of milk	(approximately 42 Gm.
Three ounces of meat or cheese	of carbohydrate, 33 Gm. of protein, 35
Two slices of bread	Gm. of fat, and
Two pats of butter.	621 calories)

Repeat in four hours.

The rationale for division of hydrocortisone between the intravenous and intramuscular routes is to assure both prompt and sustained action. Intramuscular administration of hydrocortisone is repeated in six hours if the clinical situation appears precarious. Twenty milliequivalents of potassium is given with every 100 Gm. of glucose by the same route as the glucose. Electrocardiograms are used as a guide to potassium therapy. Oxygen is used by face mask if there is cyanosis. Laryngeal spasm may require tracheotomy. If cerebral edema is a problem in spite of the 50 per cent glucose that is being given intravenously, 50 cc. of 50 per cent concentrated lyophilized human plasma may be used. Vascular shock may be treated similarly with plasma or with norepinephrine. Ventricular rates of 160 or more per minute for two hours, or cardiac arrhythmia, may be an indication for digitalization. If paralysis of the respiratory muscles ensues, artificial respiration is instituted.

COMMENT

A lesser mortality and a fuller degree of ultimate recovery have appeared to be associated with this intensive regimen for profound hypoglycemia, although, fortunately, there are no "controls" in the strict sense of the term for these cases. The problem of delayed vascular collapse has not yet been solved. Instances of irreversible hypoglycemia doubtless will still occur, but it is to be hoped that their occurrence will be less frequent with early recognition and prompt intensive treatment. Since the conditions in which profound hypoglycemia is most common are being seen with increasing frequency in practice, the management of this emergency becomes a matter of concern to all. It should, however, be emphasized that this regimen is suitable only under conditions of extreme absolute hypoglycemia and that these are invariably associated with coma: The average "insulin reaction" in the diabetic patient responds to 5 to 10 Gm. of glucose and should not be treated with excessive amounts of glucose.

*This is available as a component of some of the injectable B complex preparations.

ACKNOWLEDGMENT

I would like to express my appreciation to Dr. Stanley Pogul and his associates on the Insulin Therapy Ward of the Psychiatric Department of the Philadelphia General Hospital, with whom I have been privileged to attend current cases, to review previous cases, and to evaluate this regimen in relation to insulin coma therapy.

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Use of Antimalarial Drugs in Rheumatoid Arthritis

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THE BENEFICIAL ANTI-INFLAMMATORY action of certain antimalarial drugs has for many years been known to be effective in the treatment of "rheumatic" and certain other diseases of unknown etiology. Several investigators have reported some encouraging results in the use of quinacrine (Atabrine) and primaquine diphosphate in rheumatoid arthritis.[1-3] However, the incidence of toxicity with these two drugs was too high, and several investigators turned their attention to chloroquine (Aralen) and hydroxychloroquine (Plaquenil), less toxic 4-amino-quinoline derivatives. These two compounds had been used in lupus erythematosus, and their long-continued use in this disease revealed no serious side effects.[4-6] A number of investigators [7-9] have demonstrated that either drug may be safely used in the treatment of rheumatoid arthritis with a significant degree of success.

The exact mechanism by which the antimalarial drugs produce results in suppressing or inducing remission of the inflammatory process in rheumatoid arthritis is not known. However, in 1953 [7] the possibility was advanced that the antimalarial drugs may act by means of adenosinetriphosphatase (ATP-ase) inhibition. Earlier it was suggested that in rheumatoid arthritis the tissue requirements for ATP were increased.[10] Gold and copper, which are used in the treatment of rheumatoid arthritis, have been reported as ATP-ase inhibitors,[11] and substances of the quinine group have shown ATP sparing action.[12] These findings make the ATP-ase inhibition theory appear plausible.

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During the past several years chloroquine has been studied by many investigators for the treatment of rheumatoid arthritis. Haydu [7] treated 28 patients with rheumatoid arthritis over a six month period and found considerable improvement in 21, complete remission in 1, symptomatic improvement in 5, and no change in 1.

In a well-controlled study Freedman, in 1956,[13] reported the results of the use of chloroquine in 69 adult patients with active rheumatoid arthritis. These patients were studied by a double blind method in which half the patients received a placebo and the other half received chloroquine in daily doses of 200 to 300 mg. Moderate subjective improvement was observed in 26 of the 34 chloroquine-treated patients as opposed to 12 of the 32 controls. Moderate objective improvement was observed in 22 of the drug-treated patients and in 12 of the controls. Rinehart and others [14] reported on a study of 33 adult patients with rheumatoid arthritis, selected without regard to duration or severity of the disease. They were put at random into four treatment programs involving various sequences of the test drug and controlling agent. The authors concluded that considerably less than half of the patients improved on placebo therapy and more than half improved while taking chloroquine.

Bagnall [15,16] reported on a long-range study of 108 patients with rheumatoid arthritis and 17 patients with rheumatoid spondylitis, with observations done over a four year period, the average duration of therapy being 30 months. Chloroquine produced a successful response in 71 per cent of the 108 cases of rheumatoid

arthritis and 71 per cent of the 17 cases of spondylitis. A subjective response was rarely noted as early as 2 weeks after the beginning of therapy while an objective response was frequently seen in 6 to 12 weeks. A maximum response, according to this investigator, may require 6 to 12 months of therapy. This extensive and long-range report has been criticized [17] because of lack of controls, the short duration of the disease in the patients studied, and the administration of corticosteroids in one third of the cases at some time during the study period.

Cohen and Calkins [18] reported a double blind study on 22 adult patients with rheumatoid arthritis of at least 18 months' continuous activity. Half the patients received placebos and the other half received chloroquine in daily doses of 500 mg. After a period of 10 to 12 weeks medication was reversed, unknown to the patients. Patients were evaluated at frequent intervals as to the subjective and objective manifestations of disease activity and functional improvement. The results in this series of patients showed a significant antirheumatic activity in patients receiving chloroquine, as compared with those receiving the placebo. At least 80 per cent showed improvement; 7 patients required cessation of the medicament because of side effects. All side effects disappeared when use of the medicament was stopped. A dosage of 500 mg. was maintained throughout, irrespective of the degree of response.

Scherbel and co-workers [19] reported a comparative study of chloroquine and hydroxychloroquine and found that, while both drugs were effective agents in the treatment of rheumatoid arthritis, hydroxychloroquine was better tolerated. Fifteen of 26 patients treated with hydroxychloroquine became asymptomatic while 9 of 25 patients showed a similar response to chloroquine. Hydroxychloroquine produced fewer gastrointestinal disturbances and permitted the use of larger initial doses, as required by some patients. The better clinical tolerance and greater clinical efficacy of hydroxychloroquine led the authors to conclude that hydroxychloroquine is the preferred drug in the treatment of connective tissue disorders.

PHARMACOLOGY

Chloroquine and hydroxychloroquine are well absorbed when taken by mouth and are largely metabolized in the body, only 10 to 20 per cent being excreted in the urine.[20] After administration of 500 mg. of chloroquine phosphate, daily plasma levels gradually increase during the course of the next four weeks to approximately 0.2 mg. per liter, then reaching a plateau concentration.[20] When use of the drug is discontinued, the plasma level falls to half its plateau concentration in five days. In rats the drug was found to be concentrated primarily in the liver, kidneys, and lung, in which organs concentration of between 400 to 700 times those of plasma were observed.

TOXIC EFFECTS

Toxic reactions to the antimalarial drugs in the dosage used for antirheumatic activity vary greatly in incidence and severity. In order of toxicity, quinacrine rates highest, chloroquine intermediate, and hydroxychloroquine least toxic.[5,19] Bagnall [15] has cited toxic manifestations to chloroquine in detail and reports that 41 per cent of male patients and 64 per cent of female patients receiving 250 mg. daily showed toxic effects. It was necessary to withdraw the drug in 5 per cent of male and 15 per cent of female patients.

In general, it has been the experience of other investigators [16, 18] that approximately one quarter of the patients do not tolerate chloroquine at all, primarily due to gastrointestinal disturbances. However, it was generally found that the toxic effects of chloroquine and hydroxychloroquine were usually mild and, to date, transitory in nature, disappearing completely either on continuance or cessation of therapy or on reduction of dosage.[15, 19]

The side effects most frequently encountered were gastrointestinal disturbances, such as nausea, burning in epigastrium, vomiting, diarrhea, abdominal cramps, and anorexia.[16-18] Temporary blurring of vision, especially with use of chloroquine (due to interference with accommodation), is relatively frequent. Other observed effects were pleomorphic skin eruptions (for example, lichen, maculopapular and purpuric), all of which were generally mild and may preclude the use of an optimum dosage schedule. If the skin eruption persists on a reduced dosage or recurs after reinstitution of treatment with gradually increasing doses of the same drug, one should discontinue use of the medicament until the lesion again disappears and consider resuming treatment with another antimalarial agent.[15, 16]

Other investigators have reported hematopoietic abnormalities, notably leukopenia.[15, 16] Less frequently reported are transitory vertigo, headaches, lassitude, and neurological disturbances such as nervousness, irritability, emotional changes, and nightmare.

Generally, manifestations of intolerance to one antimalarial compound need not imply intolerance to others. Reduction of dosage or change to a different antimalarial agent will often allow the reactions to disappear. Subsequent return to the original compound may then be tolerated without the recurrence of the original reaction.

All instances of side reactions reported to date have subsided with cessation of therapy.

DOSAGE

Antimalarial compounds are cumulative in action and require weeks to exert their beneficial therapeutic effects, whereas numerous side effects may occur early. Since there is a great individual variation in patient response, a rigid dosage schedule cannot be followed.

The initial dosage of chloroquine phosphate in the usual adult is 250 to 500 mg. daily, given orally, a single or divided dose taken with meals or frequently as a single dose at the evening meal.[15, 18] With hydroxychloroquine, the usual daily starting dose range is from 400 to 600 mg. orally, with a minimum of 200 mg. and a maximum of 1 Gm. daily.[18]

In a small percentage of patients, troublesome side effects may require temporary reduction of the initial dosage. Later (usually 5 to 10 days) the dose may be gradually increased to the optimum response level, often without the recurrence of the side effects.

The maintenance dosage cannot be determined before at least 4 to 12 weeks of treatment with the initial dosage schedule in order that the effect be properly evaluated. If a good response is obtained, the dose is reduced by 50 per cent and continued at a usual level of 250 mg. chloroquine daily or every other day. With hydroxychloroquine, the usual maintenance dose is 200 to 400 mg. daily.

If use of the medicament is withdrawn, a relapse, if it occurs, will usually be manifested within 3 to 12 months. Resumption of therapy, as mentioned, will again be found effective.

COMMENT

From evidence reported by the various afore-mentioned investigators the antimalarial compounds, namely chloroquine and hydroxychloroquine, appear to have significant antirheumatic action in some patients. However, in rheumatoid arthritis, a disease that exhibits a variable course in different individuals and spontaneous remissions, the evaluation of any agent is difficult. This is particularly true when an agent like chloroquine requires as long as weeks or months to take effect. More controlled investigation and observation is necessary before a significant antirheumatic activity is established as a fact in this group of compounds. It is also possible that the toxic reactions of patients to these drugs may prevent their widespread use in rheumatoid arthritis.

A newer antimalarial agent also being investigated is amodiaquin (Camoquin).

The antimalarial drugs must be regarded as still in the investigative stage. However, if they are proved to have definite antirheumatic activity, they will be a welcome addition to the therapy of rheumatoid arthritis. It is possible that a combination of therapy, of antimalarials, salicylates, gold salts, and the steroids, may prove to be efficacious, especially at certain stages of the disease.

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Statistics on Blindness

Remarkable progress has been made in reducing the number of new cases of blindness among children, according to the statisticians of the Metropolitan Life Insurance Company. Not only has blindness due to infectious diseases decreased sharply among school-age children but loss of sight due to the administering of oxygen in high concentration to premature infants has become rare.

Other details from the statisticians' report include the following: 1. Persons in the United States who are either totally blind or have impairment of vision sufficiently great to prevent normal activities number 345,000, a rate of 2 per 1,000 of the population. New cases are estimated at 30,000 yearly, or 17 per 100,000. 2. Slightly more than half of the blind are 65 years of age or over; approximately 30 per cent are between ages 40 and 64 years; 12 per cent are between 20 and 39 years; and 7 per cent are under age 20. 3. The most frequent causes of blindness very generally have their onset in middle and later life and include specific eye conditions of unknown etiology, particularly glaucoma and cataract, and such general disorders as arteriosclerosis, high blood pressure, nephritis, and diabetes. 4. Men have a higher blindness rate than women, resulting in part from the higher incidence among men of blindness due to accidents and to the earlier development of arteriosclerotic and other degenerative changes.

Much can be done to prevent blindness. "A study in Baltimore showed that half the cases of blindness could have been prevented," the report states. "Early diagnosis and treatment of pathological eye conditions are the best available means to conserve sight. Periodic health checks, particularly among people past middle life, should include examination of the eyes for glaucoma and cataract, as well as for changes due to degenerative vascular diseases."

Placenta Praevia

Michael Marie O'Brien, M.D.

THE PLACENTA NORMALLY is located upon the anterior or posterior wall of the uterus; it is round or discoid, 15 to 20 cm. in diameter, 1.5 to 2 cm. thick, and about one-sixth the weight of the fetus.[1] However, in some cases, the placenta is implanted upon the lower uterine segment and overlaps the internal os; in such a position, it is one of the most common causes of hemorrhage during the last half of pregnancy.[2] Schmitz [3] attributes 33 per cent of ante-partum bleeding to this obstetric complication of placenta praevia.

In 1949 a group of obstetricians decided on a method of classifying the various types of placenta praevia, in order that there might be uniformity in reporting the incidence of this entity.[3] Three types are recognized: (1) total (the internal os is completely covered by the placenta), (2) partial (the internal os is partially covered), and (3) low-implantation (the placenta does not extend beyond the margin of the os but can be palpated upon cervical examination).

The incidence of placenta praevia varies according to the type of hospital reporting any particular series; those hospitals that receive obstetric problems from large areas, such as the Mayo Clinic, have a greater incidence. Mayo reports 1 in 89 cases; Eastman [1] says that his figures show 1 in 200; Johnson [4] shows 1 in 250; and 1 in 225 is reported by Reich.[5] Our particular series, covering the years 1952 through 1956 at Woman's Medical College Hospital, involves 43 cases in 6,101 births, an incidence of 1 in 142.

The incidence of each type is reported, but one must understand that the classification is relative: The types change during the process of dilatation and what would be called total on one examination may be partial later in the course of labor.[1] Many series still report the type of placenta praevia as marginal and low-lying instead of as low-implantation. Many of the cases reported in our series are labeled in such a way; in order to be consistent, I have added these types together and they are reported in this paper as "low-implantation" cases. This may account for some of the discrepancies noted between our figures and those of others. We have an incidence of 80.6 per cent low-implantation cases; Eastman reports 46 per cent (table I). In all series low-implantation is the type most frequently reported.

The etiology of placenta praevia is not known definitely; however, the factors thought to favor its occurrence are: multiparity, atrophic changes in the endometrium, and an embryologic factor.[1] Hoopes [6] explains this last factor as follows: ". . . the primary implantation in the lower uterine segment is associated with such extensive cleavage of the decidua vera that the extension of the placenta to the region of the internal os is facilitated."

All series checked for this paper reported a very high incidence of placenta praevia in multiparas: 86.1 per cent of our cases, 89.7 per cent of Reich's [5] (Bellevue), and 88.5 per cent of Eadie's [2] (table II). Some authors report that the incidence of this type of implantation increases with the parity of the patient; one author states that 45 per cent of his patients had 5 or more children, and 12.9 per cent of our cases fell in this group. Our records show that the greatest incidence (63.8 per cent) occurred in patients who had 3 or 4 children. Hoopes [6] quotes 4.2 children as the average, with 32.9 years being the average age of the patients. About 61.1 per cent of our patients were under the age of 30 years.

The atrophic changes in the endometrium were not reported statistically. However, it is

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TABLE I
Types of Placenta Praevia

	Woman's Medical College Hospital	Bellevue	Eadie
Total births	6,101	69,815	26,136
Incidence of placenta praevia	1:142	1:225	1:200
Type, %			
Total	11.1	27.4	24.5
Partial	8.3	31.6	20.6
Low-implantation	80.6	35.2	54.9

thought that the defective vascularity of the decidua in these endometria is such that the placenta must extend widely and thinly over the endometrium in order to obtain enough nourishment for the growing fetus. This factor is thought to be responsible for the high incidence of abortions in patients with placenta praevia.[6] In our series 27.7 per cent of the patients had a history of previous abortions, but whether these were due to placenta praevia could not be ascertained. Hertig and Livingston [7] implicate the low-implantation type of placenta praevia in 5.6 per cent of abortions studied by them. Many abortions caused by placenta praevia are not seen because the fetus is expelled before the etiology of the bleeding is recognized.

Age and race seem to be unrelated to the incidence of this obstetric problem and both are ignored in all the series.[8, 9]

The classical symptom of placenta praevia is painless hemorrhage in the last trimester of pregnancy. Eastman [1] lists five symptoms associated with this entity: (1) painless hemorrhage after the seventh month, (2) abortions in the third month, (3) small initial bleeding with later recurrence or continuous bleeding in small amounts, (4) bleeding only at the time of delivery, and (5) abnormal presentations.

In our series painless hemorrhage in the last trimester was the presenting symptom in 69.3 per cent of the cases. Only 13 per cent had bleeding in the first and second trimesters. For the most part, the initial hemorrhage was small, however, in 16.6 per cent of the patients it was excessive and necessitated immediate delivery. The second episodes were most profuse, and, with vaginal delivery, bleeding during the delivery was also usually profuse. The experience at our hospital seems to substantiate Johnson's [4] observation that the first hemorrhage is usually small and nonfatal. Reich [5] and Eadie [2] agree with Johnson; Eastman [1] notes that in one series 14 out of 41 patients were hospitalized for bleeding, with the amount of blood lost being relatively small. In our series 16.6 per cent of the patients were hospitalized for a short period of time after bleeding episodes. Most of these went on to complete the pregnancy.

TABLE II
Rates of Placenta Praevia in Primiparas and Multiparas

Parity	Woman's Medical College Hospital, %	Bellevue, %	Eadie, %
Primipara	13.8	10.3	11.5
Multipara	86.1	89.7	88.5
Multipara, 5 or more	12.9	45.0	—

Abnormal presentations are more frequent in patients with placenta praevia because, according to Eastman,[1] the placenta interferes with the accommodation of the fetal head. In his series 30 per cent of the patients had abnormal presentations; Schmitz [3] reports 30 per cent also. We had 24.9 per cent abnormal presentations: 13.8 per cent breech and 11.1 per cent transverse. In table III, I have compared our figures on presentation with those reported by Reich [5] at Bellevue Hospital. When we read that only 5.1 per cent of the general obstetric population have abnormal presentations, we can see that there is an unusually high incidence of these presentations in patients with placenta praevia.

TABLE III
Types of Presentation in Patients with Placenta Praevia

Presentation	Woman's Medical College Hospital, %	Bellevue, %
Vertex	77.7	73.2
Breech	13.8	14.2
Transverse	11.1	11.5

To make the diagnosis of placenta praevia, one must rely mostly on the clinical history. Abdominal, roentgenographic, and pelvic examinations are used to confirm the diagnosis. Many obstetricians think that a history of ante-partum bleeding is sufficient. Others prefer to use roentgenographic examinations as an aid. Soft-tissue studies, cystograms, and indirect placentography are some of the techniques used. The soft-tissue technique is preferred by many. It has some limitations in that hydramnios and multiple pregnancy conditions obscure the placenta. In cystography an opaque material or air is inserted into the bladder. If the head is separated from the bladder by a distance of 1 cm., the diagnosis of placenta praevia can be made. Indirect

placentograms are thought to be more accurate; some say 95 per cent accurate. Placentography is done with the patient in the erect and recumbent positions. The displacement of the fetal head from the midsagittal or midcoronal line is indicative of placenta praevia, provided a full bladder, full rectum, and neoplasm can be ruled out. Schmitz [3] does not believe that roentgenographic diagnosis is reliable. On the other hand, Eadie [2] recommends the use of the air cystogram or the soft-tissue study in making the diagnosis. In our series the use of a roentgenographic study is mentioned only once.

To make a definite diagnosis, a sterile cervical examination should be done. However, this procedure is very dangerous and is attended in many cases by serious hemorrhage. For this reason, it is recommended by most obstetricians only when one is ready to deliver the fetus. Even rectal examinations are frowned upon. When the time for delivery has come, the patient is taken to the operating room where a double setup is ready. This means that one is prepared to do either a vaginal or an abdominal delivery. Blood is on hand in case severe bleeding occurs. Eastman [1] states that fatalities from placenta praevia occurred only when vaginal examinations were made without these precautions. Johnson [4] says that a sterile vaginal examination can be made early in the pregnancy, provided it is made in the operating room with the afore-mentioned precautions. Delivery need not be done at that time. Johnson feels that a careful examination will not cause any bleeding. The diagnosis is then definitely made and the patient can be watched until ready for delivery.[4] In our series Eastman's recommendations were followed for the most part. In 2 cases a sterile vaginal examination was done at the time of labor, but in these cases the patient had no symptoms indicating the presence of placenta praevia.

The prognosis in this obstetric complication is excellent for the mother. No maternal deaths occurred in our series, which covered a five year span. Before 1943 there was a maternal mortality of 2.3 per cent associated with this condition. Eadie [2] believes that the reduction since that time is due to the use of antibiotics and blood transfusions. The lower uterine segment and cervix in these patients are friable and susceptible to infection and bleeding. With the control of infection by antibiotics and of shock by blood, the mortality rate today is practically zero. MacAfee [10] reports 1 death in 173 cases; Charity Hospital [8] had only 1 maternal death due to placenta praevia out of a total of 131 maternal deaths from 1946 through 1955, with a total of 104,703 live births.

The outlook for the child is not so good. In New York City, placenta praevia accounted for 22.9 per cent of perinatal deaths for 1954,[11] including both fetal and neonatal deaths. Reich [5] reports that fetal mortality has dropped at Bellevue Hospital from 28.4 per cent during the years 1919 to 1941 to 13.8 per cent in the period 1941 to 1954. These figures are for deaths associated with placenta praevia. Reich attributes this drop to the newer methods of handling patients with placenta praevia. In our series there was a fetal mortality rate of 25 per cent, a figure consistent with those reported by others. Schmitz [3] reports 23 per cent and Eadie [2] reports 43.2 per cent, while Grant [12] in England had a fetal loss of 16 per cent.

Prematurity is listed as the main cause of death in all series. For that reason, Johnson [4] and MacAfee [10] are proponents of the idea of watchful waiting; that is, they do not terminate the pregnancy when the diagnosis of placenta praevia is made if it is possible to maintain the life of the mother. They prefer to keep the patient under observation and deliver the child when the fetus has reached the age of 36 weeks. Since the initial bleeding is not fatal, they see no reason for action unless the life of the mother is in immediate danger. In our series, with prematurity being the most frequent cause of death, the greatest number of deaths occurred in that group of fetuses with a birth weight of less than 1,600 Gm. (about 3.5 lb.). Fifty per cent of all babies in this series were premature; of this 50 per cent, only 50 per cent survived. Only 2 infants with a gestational period of less than 30 weeks lived; the others were 35 weeks or older.

Since 1945, when Johnson and MacAfee, working separately, instituted the method of watchful waiting, the treatment of placenta praevia has undergone changes. Prior to that time it was customary to deliver the patient as soon as the diagnosis was made. Many patients were delivered vaginally, which resulted in profuse hemorrhage or serious infection. The fetal mortality was high due to the premature delivery of the infant. Johnson and MacAfee advocate trying to keep the fetus in utero until it is able to survive outside. At 36 to 37 weeks' gestation the fetus is consid-

ered ready for delivery and a cesarean section is performed. If labor starts and is not accompanied by excessive bleeding, it is permitted to continue with no effort to hasten it. The membranes are ruptured to allow the head to descend and to act as a tampon on the placenta. This procedure reduces the intrauterine volume and gives the sinuses a chance to be retracted. Oxytocin is not used, as it is thought that slow labor, with long intervals between contractions, helps to cut down the amount of blood lost. Bleeding occurs with dilatation and effacement as the placenta separates from the lower segment of the uterus; the long intervals allow the separated sinuses to retract. Once the fetus is delivered the placenta separates completely from the uterine wall with no manual assistance needed. Johnson [4] insists that manual delivery of the placenta leads to excessive blood loss, as the abrupt removal of the placenta from the lower uterine segment causes considerable bleeding and as this section of the uterus is physiologically unable to retract as rapidly and strongly as the upper segment. Slow removal allows the sinuses time to be retracted gradually. Both Johnson and MacAfee suggest that blood be ready at the delivery.

Hoopes [6] states that abdominal delivery is the preferred method, particularly in cases of marginal placenta in which labor has not started and in all cases of partial or total placenta praevia. He states that vaginal delivery is associated with a greater risk of cervical and lower uterine segment lacerations. Abdominal delivery enables the physician to visualize the site of the placenta and to control the bleeding sinuses more easily. However, each case must be studied and treated according to individual needs.

Reich [5] agrees with Hoopes [6] and states that cesarean section is preferred for all cases except in special circumstances such as early labor, error in diagnosis, or presence of advanced labor at the time of admission. In all cases adequate blood is to be ready. In his series he notes that, during the years 1919 to 1941, 81.6 per cent were delivered vaginally with special procedures such as internal version, the Voorhees bag, induction of labor, and vaginal packing. With these procedures there was a fetal mortality of 44.4 per cent while among those delivered by cesarean section there was a fetal mortality rate of 12.5 per cent. In his second series, covering the years 1941 to 1954, 84.6 per cent were delivered by cesarean section, with a fetal mortality rate of 10.1 per cent; among those delivered vaginally there was a fetal mortality rate of 17.6 per cent. Reich attributes this marked decrease to use of cesarean section in patients who carried the fetus beyond 36 weeks. He notes that rectal and vaginal examinations were not done and that every effort was made to avoid hemorrhage. In cases where there was massive hemorrhage, the patient was delivered immediately by cesarean section. Grant [12] waits until the thirty-seventh week and then delivers the fetus by amniotomy, with or without Willett's forceps, or by section.

Eastman [1] suggests the following procedure for a patient with placenta praevia: 1. Hospitalize the patient but do not make rectal or vaginal examinations. Watch the patient and give transfusions if necessary. If bleeding stops, the patient may be sent home until ready for delivery. 2. No packing or digital examination is to be done in any patient who bleeds. Sterile vaginal examinations may be done in the operating room with the double setup and blood ready. 3. Delivery may be vaginal or by cesarean section.

For vaginal delivery Eastman lists the following methods to be employed. The membranes are ruptured to allow the head to settle against the placenta. If bleeding persists, Willett's forceps are applied to the scalp of the infant and a pound weight is attached to the forceps. This provides traction on the head and puts additional pressure on the placenta. This should not be used in primiparas, in partial or total placenta praevia, or in cases where there has been previous hemorrhage. The Voorhees bag may be inserted to act as a tampon, but this has disadvantages. The placenta, cervix, and lower uterine segment are traumatized by the bag; there is profuse bleeding between the expulsion of the bag and delivery. If it is expelled before complete dilatation, extensive lacerations result. Hicks' version, with delivery of one foot to allow the fetal buttocks and thigh to rest against the placenta, is not recommended, since a high fetal mortality rate is associated with this procedure.

Eastman prefers cesarean section, as this method avoids the lacerations that occur in the friable cervix and lower uterine segment and also shortens the ante-partum bleeding.

In the selection of a method certain factors must be considered: 1. Primiparas, patients with total or partial placenta praevia, and patients with massive hemorrhage should be

delivered by cesarean section. 2. In the presence of infection or in a multipara with a dilatation of 4 cm., vaginal delivery should be used. In every case the use of blood is the most important part of the treatment, as it is necessary to maintain the blood volume at normal levels. There is agreement among all on the main points of treatment.

In our series 61.1 per cent of the patients were delivered vaginally and 38.9 per cent were delivered by cesarean section. Those delivered vaginally were admitted while in active labor that was progresssing rapidly. Fourteen per cent had been under observation and were delivered when the fetus was more than 36 weeks old. The Voorhees bag was used once, in a multipara with a double footling presentation. This patient had been admitted with ruptured membranes and bled during labor. A podalic version was done in 1 case where there was polyhydramnios, a dilated cervix, and a floating head. Transfusions were used in 27.7 per cent of the patients, and only 11.1 per cent had anemia as a result of ante- or postpartum bleeding.

Our results compare favorably with those of the other series. However, in recent years, the advisability of doing so many cesarean sections has been questioned. One author believes that this method of delivery is associated with a high maternal morbidity and is one factor favoring fetal mortality. He suggests that this problem be investigated further.

SUMMARY

In this paper the various types of placenta praevia, the possible etiology or factors favoring its occurrence, the methods of diagnosis, and the methods of treatment are discussed. Our records on all these points were compared with those reported by others all over the country and were found to be more or less in agreement on all points.

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"Terra Incognita" in Medicine*

Catharine Macfarlane, M.D., F.A.C.S.

I AM VERY GLAD to be with you this evening to meet the new students and the returning students as well. I hope you all like maps. I saw a very interesting old one not long ago of Africa. A few countries were drawn in around the edges of the continent, but the center was empty save for two words in large letters. The words were "terra incognita," unknown territory, a very significant phrase.

A similar map might well have been used to illustrate the state of medical knowledge on that September day in 1895 when I was welcomed to the Woman's Medical College of Pennsylvania, as you are being welcomed today. At that time there was a great deal of terra incognita in medicine. In that year a German scientist, Conrad Roentgen, discovered the x-ray. Until then, the time-honored procedures of inspection, palpation, percussion, and auscultation had been the only methods available for finding out what was inside a human body. Soon timid and tentative attempts were begun to make this new discovery available for the diagnosis of disease. If the roentgen-ray showed shadows of keys and rings, perhaps it would throw shadows of bones and other structures. As you all know, it did.

Three years later, my graduation year, Pierre and Marie Curie, stirring pitchblende in a shack on the grounds of the University of Paris, succeeded in isolating a new element—radium. Soon this marvelous substance was tried out timidly and tentatively in the Vienna Frauenklinik for the treatment of inoperable cancer of the uterus. Many patients for whom previously the only alternatives had been morphine and death were granted many years of life and comfort.

Also in 1898 a vessel of the U.S. Navy, the battleship Maine, was sunk in Havana Harbor. As a result the United States entered into war with Spain. Young men on both sides of the conflict became sick and died of typhoid fever because typhoid vaccine had not yet been discovered.

It was not until 1902 that Major Walter Reed and his U.S. Army associates established the role of the *Aedes aegypti* mosquito in transmitting yellow fever and proved that this disease could be prevented by mosquito control.

Think of the hundreds of deaths from pneumonia that have occurred in your own lifetime because the sulfonamides had not yet been discovered, or think of the deaths that have occurred from septic infection because Sir Archibald Fleming had not yet observed a spot of mold interfering with the growth of a staphylococcus culture. All of you have seen children and grownups on crutches or in wheel chairs, paralyzed from poliomyelitis, because Dr. Jonas Salk of Pittsburgh did not discover poliomyelitis vaccine until 1954. And, getting back to Africa, Dr. Louise Pearce, a member of the Board of Corporators of this College, received a decoration and an award from the Belgian Government in recognition of what she and her associates of the Rockefeller Institute had done in the control of sleeping sickness in the Belgian Congo.

Thus the area of our medical terra incognita grows smaller every year, although it has not disappeared. There is still much more to be done, and you young people will be here to do it. We still have the common cold, resulting in millions of lost work hours every year. We still have cancer, which took a toll of 250,000 lives in this country in 1956. We still have tuberculosis, arteriosclerosis, coronary heart disease, cerebral hemorrhage, and arthritis.

Somebody must find out how and why individuals get these diseases and what to do about them. Why should that somebody not be one of you? Since shooting at the moon has

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become an approved scientific pursuit, it is certainly permissible for you fortunate young people today to say "the sky is the limit."

As a clinician, having paid my full measure of respect to the laboratory workers, I feel obliged to remind you that medicine is an art as well as a science. All of you will eventually learn that the sick man, woman, or child is not merely a collection of symptoms recorded in the intern's history or on the nurse's chart. Each is a bundle of hopes and fears. In addition to drugs or surgery, each one needs faith and hope and love. The faith may be in miracle drugs, in a revered physician, or in God, probably in all three. The hope must never be quenched. There must always be some new drug or some new treatment to try or there must be one tomorrow. The courage of one patient after a radical operation for cancer was strengthened by daily visits from her pastor, the courage of another by daily visits from a devoted husband and friends. You and I need the help of a higher power in our efforts to heal God's children.

On Sunday, Aug. 17, the Philadelphia and New York papers recorded the passing of a great man, Dr. Chevalier Jackson. Among many other achievements, he developed the bronchoscope and taught many pupils how to use it. He was thus instrumental in saving the lives of thousands of children throughout the world. Dr. Jackson was a great friend of women physicians. In Pittsburgh he trained a graduate of this College, Dr. Ellen J. Patterson. In Philadelphia he trained another graduate of this College, Dr. Emily Lois Van Loon, now our Professor of Otolaryngology. From 1935 to 1941 Dr. Jackson was president of this College. One September afternoon, in this very room, I heard him give the welcoming address to the student body, just as I have been privileged to do this evening. I remember his closing words, "The Woman's Medical College is here to help you. To help you as undergraduates, to help you after you graduate. If you are ever in trouble, professional or otherwise, do not hesitate to call on the Woman's Medical College. Corporators, faculty, personnel, and I personally stand ready to help you."

This evening it is my privilege to echo the words of this great man. Good luck and God bless you.

Progress in Hospital Planning and Construction

The number of people in the United States without ready access to general hospitals has dropped from 10 million to 2.8 million since 1948, according to the Public Health Service. Even in the most rural areas only a small percentage of the population is now without nearby hospital facilities. This and other evidences of progress in hospital planning and construction, as well as needs for other types of health facilities, are shown in a new publication, "The Nation's Health Facilities—Ten Years of the Hill-Burton Hospital and Medical Facilities Program, 1946-1956," which includes a summary of the program to Jan. 1, 1958.

During the first 10 years of the program, 3,047 projects were approved for construction. In addition to general hospitals, these new health facilities include nursing homes, diagnostic and treatment centers, rehabilitation facilities, public health centers, and state health laboratories. Of the total cost of 2.5 billion dollars, the state and local share was nearly 1.7 billion dollars. The remainder was provided by the Federal Government. States with the greatest need and the lowest income have received the most Federal funds per person.

The report reviews, as of July, 1956, the status of each type of non-Federal health facility reported by the states in the plans approved under the hospital survey and construction program. It includes new analytical data, particularly for general hospitals in metropolitan areas, general hospitals in the 375 hospital regions, and psychiatric units in general hospitals. The publication is sold by the Superintendent of Documents, Government Printing Office, Washington 25, D. C., for $1.25.

The Woman's Medical College of Pennsylvania

SINCE ITS FOUNDING in 1850 the Woman's Medical College of Pennsylvania has furnished an opportunity for medical education to women. Begun in a day when, except for Elizabeth and Emily Blackwell, women had not been admitted to medical schools, it has continued to the present time to carry out the purpose for which it was founded.

Although today in the United States only one four-year school and one two-year school do not admit women, the number of applicants for Woman's Medical College continues to be large enough to allow a choice of qualified applicants for the 50 places in the entering class.

The classes are small enough to make individual teaching possible but large enough to furnish stimulation and lively discussion.

The student laboratories are well equipped. Opportunities for research are given the students, as there are a generous number of student research fellowships. A new wing devoted primarily to research facilities has just been started and will be in use by 1961. One floor of this wing will house enlarged quarters for the physiological chemistry department and will make possible the acceptance of larger entering classes.

The student spends the clinical years in the College Hospital and in other hospitals in Philadelphia—Philadelphia General, the Veterans Administration Hospital, and various specialty hospitals such as the Eastern Pennsylvania Psychiatric Institute.

The student body is an interesting and cosmopolitan group. Preference is given, of course, to Pennsylvania applicants, but students from all over the United States, Canada, and Latin America are enrolled. Countries much farther away are represented—India, China, and Greece—and even a student from Afghanistan has studied at the College.

The schedule in a medical school is demanding and requires long hours of study. The students, however, do take advantage of the opportunities that Philadelphia offers them in music, art, and the theater. Some of them combine matrimony with medicine very successfully before and after graduation.

After graduation the opportunities for internships are many. Residencies in various specialties are open to women in teaching centers and good hospitals all over the country. And there are many physicians needed for general practice, which gives such personal satisfaction to the physician and to the patient. The need for physicians is increasing with population growth. The Woman's Medical College is happy to help meet that need.

Second Century of Building Progress for The Woman's Medical College of Pennsylvania

DEC. 9, 1958, WAS AN IMPORTANT day in the annals of the Woman's Medical College, for it marked the ground-breaking of the new 2 million dollar Research Wing, which will be completed early in 1961.

This new wing will provide laboratories and other facilities for the College's expanding research program. One entire floor of the new building, reserved for teaching, will permit an increased enrollment of 20 per cent in the student body. The renovation of laboratories in the main building will provide additional space for diagnosis and treatment of patients. Corridors on each floor will connect the Research Wing with the College and the Hospital.

The actual ground-breaking was uniquely performed by 12 professors who manned twin shovels. They are the professors whose departments will be located in this new five story brick building.

Architect's drawing of the new 2 million dollar Research Wing for Woman's Medical College.

Dean Marion Fay, in expressing thanks to the faculty members, said: "These teachers, without whom bricks and mortar, however beautiful, could never make a school, set an example every day of what a scientist and a physician should be. This is a day of rejoicing for all of them who have worked long in cramped and inadequate quarters—a day when we can thank them for all they have done for the Woman's Medical College, for science, for medicine, and for medical education."

Dr. Catharine Macfarlane and Mrs. John B. Kelly both expressed appreciation to the College public and to the many friends and alumnae who have made the building fund possible. Dr. Macfarlane spoke of the great medical advances made possible by the laboratory work of pioneers and said, "I have faith that some day some genius in a laboratory will discover the cause of cancer, perhaps in this very building for which ground is being broken today." She concluded by quoting Field Marshal Jan Smuts who once said, "Science is perhaps the clearest revelation of God in our day."

This new research wing is the third major expansion undertaken by the College since it attained centennial status in 1950. Ann Preston Hall, a modern nurses' residence and school of nursing, was completed in 1952 at a cost of $742,000; in 1954 a preventive medicine wing, costing $146,518, was added to the College; and renovations in the Hospital in 1952 provided 42 more beds at a cost of $166,000.

The present wing was made possible by a grant of $500,000 from the Federal Government, which earmarked it for research facilities. Contributions from charitable foundations, industry, and friends of the institution, including its very active alumnae, auxiliary members, and National Board, have oversubscribed the matching funds so that now only $700,000 is needed to complete the fund.

The expansion program for the College includes for the near future a new library, an auditorium, and a much-needed addition to the Hospital. The library will include a section for the housing of the Library of the American Medical Women's Association, for which funds have been most generously contributed by members and friends of the AMWA. The College is custodian of this library collection. Due to the lack of space in the present College library, the books and pamphlets have not yet been catalogued or placed on the shelves.

Dr. Roberta Hafkesbring and Dr. Ruth Miller break ground for the new research wing in which their departments will be located. Waiting her turn at left near Dean Marion Fay is Dr. Mary McKee Porter.

President's Message

Since the March issue is the special number of the Woman's Medical College, it seems fitting to use this as my theme. Remembering that the College was founded in 1850 and that it has endured through many storms and upheavals, this institution for the medical education of women stands as firmly as the Rock of Gibraltar.

To one who attended, upheld, and profited by coeducation, the question arises as to what are the special values of separate medical schools for women. One answer may be found in what a dean of women pointed out to a group of undergraduate students participating in extracurricular activities. She showed them that it was beneficial for young women of college age to work together for a given project. While she did not discourage participation in organizations with men or in social life with them, she felt it of value for them to learn to understand and co-operate with one another. I believe she was right, but perhaps those who had the opportunity for such experiences in a coeducational institution were exceptional. The Woman's Medical College does provide this kind of experience for all young women going into the profession of medicine.

Futhermore, nowhere except in a college staffed primarily by women, or in the hospital co-ordinating with it, are there as many openings for professorships or heads of departments. Women need to have their skills recognized when they are equivalent to those of men, in a similar manner as our male colleagues. This sense of achievement, with its accompanying prestige, is strenthening to one's ego.

In much the same way women can find values in pooling their interests, time, and abilities in working for the American Medical Women's Association, which provides similar opportunites of working together for graduates of the Woman's Medical College, as well as of all other recognized medical schools. There are rewards in earnest effort.

Once, the import of the phrase, "The reward for good work is more work," was objectionable to me, but now I recognize and accept the truth of this statement. Ralph Waldo Emerson provides us with a somewhat similar thought in a more profound way in this saying: "I look on that man as happy who, when there is question of success, looks into his work for a reply."

There seems to be no question of success for the Woman's Medical College, now that it is embarking on a development program, with its new wing for research. We are fortunate to have such an outstanding woman physician as Dr. Catharine Macfarlane be chairman of the Woman's Medical College Committee, which Committee serves as a liaison between the College and the AMWA.

As president of the AMWA I feel honored to be an ex-officio member of the Board of Directors of the Woman's Medical College of Pennsylvania and send most cordial greetings.

Katharine W. Wright

American Medical Women's Association, Inc.

TENTATIVE PROGRAM OF 1959 ANNUAL MEETING

Sheraton-Ritz Carlton Hotel, Atlantic City, N.J. *June 4-7, 1959*

Thursday, June 4

Committee Meetings

Executive

Publications

Finance

Others as called by the chairmen

Friday, June 5

9:00 a.m.—Official convening of 44th Annual Meeting. General business session

12:00 noon—All resolutions in the hands of Reference Committee "A." Moderators: Dr. Rosa Lee Nemir and Dr. Katharine Boucot

12:30 p.m.—Luncheon: Panel on "The Development of Physicians as Advisers"

6:30 p.m.—Hospitality hour: Branch Four, New Jersey, hostess

7:30 p.m.—Woolley Memorial Lecture: "The Diagnostic Approach to Behavior Problems in Children," Dr. Ruth Bakwin.

Saturday, June 6

9:00 a.m.—General business session

11:00 a.m.—Reading of resolutions

12:30 p.m.—Luncheon: "Preceptorships" (speaker to be announced): Dr. Mary K. Helz, Chairman of Medical Education, presiding

1:45 p.m.—Workshops based on Friday's panel material

3:00 p.m.—Reports from workshops: Leader of each summarizing

6:30 p.m.—Hospitality hour: Mead Johnson and Company, hosts

7:30 p.m.—Inaugural banquet: Dr. Jessie L. Brodie, inaugural address

Sunday, June 7

9:00 a.m.—General business session: New business

12:30 p.m.—Luncheon: Conclusions drawn from panel and workshops. Adjournment

2:00 p.m.—Program planning meeting, new committee chairmen

4:00 p.m.—Meeting of new Executive Committee and new Board of Directors

6:30 p.m.—Buffet supper for all women physicians attending AMWA and AMA meetings. Hostess: Mrs. Agnes D. Beaton, Director, Women's Acttivities, Automotive Safety Foundation

For room reservations, see page 209; for meal reservations, page 221.

Of Special Interest

NATIONAL ORGANIZATIONS AND HEALTH

Dr. Katharine W. Wright, President of AMWA, represented the Association at a conference of National Organizations and Representatives, arranged by the U.S. Department of Health, Education, and Welfare in Washington, D.C., Dec. 10. The agenda for the meeting included statements on the "Purpose of the Conference," by the Secretary of HEW, Arthur S. Fleming; "Public Health Service Activities," by the Surgeon General, Dr. Leroy E. Burney; and "Children's Bureau Activities," by Dr. Katherine Bain, Deputy Chief of the Children's Bureau.

Later, discussion groups considered the joint responsibilities of the Department of HEW and of national organizations on the following: (1) emerging health trends and problems of national importance, including research and manpower needs, methods of achieving rapid application of knowledge for disease control, and health information and education needs; and (2) suggestions for more fruitful relationships between the Department and national organizations.

Dr. Burney stated, "Our basic responsibility, as a principal Federal health agency, is to protect and promote the health of the people of the United States. We discharge this responsibility by providing nationwide leadership in health, and through direct operation of technical assistance and grants in research, training, health facilities, and disease control. Over the years, the interest of the Committee (on Appropriations) and Congress as a whole has enabled us to carry out these responsibilities and to meet new health problems.

"Our Nation's response to the enormous challenge of today's world depends very largely, I believe, on the strength and vitality of our people. No matter how advanced we become technologically, our real defense is in the people. We must, therefore, continue our efforts to maintain and extend high levels of health in this country."

"WHO'S WHO OF AMERICAN WOMEN"

To answer the need for "a book confined to the woman who stands out from her sisters," the publishers of "Who's Who" have recently announced the first edition of the new biennial publication, "Who's Who of American Women." The first volume, which includes biographies of almost 20,000 outstanding American women, emphasizes the qualitative and quantitative importance of women in modern life in a separate vocational-geographical index accompanied by statistics. In terms of professional groups, women physcians and social scientists share the rank of fifth largest group. With 931 entries, each of these professions comprises 4.9 per cent of the total. The biggest vocational category is that of women who are primarily club, civic, and lay religious leaders, nearly all at the big city or higher levels. Approximately 2,983 (15.7 per cent) of the entries are in this group. Businesswomen, nearly 2,500, are the second largest group, followed by college educators (1,850) and artists (1,273).

COMMEMORATIVE STAMPS

A memorial stamp commemorating the hundred and fiftieth anniversary of the first successful oophorectomy on record is being sought by the House of Delegates of the AMA. The operation was performed by Dr. Ephraim McDowell on Jane Todd Crawford in Danville, Ky., on Dec. 25, 1809.

In Canada the Associated Country Women of the World will be honored by the issuing of a commemorative stamp in May. The concept of the organization originated with a Canadian woman, and the idea became a reality in 1933 at a conference in Stockholm, Sweden. Today the Association, dedicated to the betterment of all mankind, has membership in more than 30 countries.

NEW PSYCHIATRIC HOSPITAL

Gracie Square Hospital in New York City, a private psychiatric institution with facilities for 232 bed patients, admitted its first patients early in February, 1959. The Hospital includes provision for admissions on an emergency basis, for medical and surgical care in combination with psychiatric treatment, and for special patients such as geriatric patients, addicts, and those with neurological and neurosurgical complications. A "day hospital-night hospital" system has been developed for part-time pa-

tients: Some patients return home in the evenings and over weekends; some pursue work or other outside activities during the day but receive intensive treatment at the Hospital each evening. The Hospital's stated policy is to maintain close co-operation with the referring physician of each patient, and referring psychiatrists who are members of the American Psychiatric Association may visit their patients at any time.

SYMPOSIUM ON POLIOMYELITIS

Dr. Jonas E. Salk, speaking at a symposium titled "Polio and Beyond" at the University of Michigan in Ann Arbor, emphasized that the fourth shot now being recommended of the vaccine that bears his name is necessary only until the potency of commercially manufactured vaccine is increased to the level that he achieved in his laboratory. He presented a two part program for dealing with the present situation: 1. Give a fourth injection of the vaccine presently available to compensate for deficiences in protection which sometimes remain after three injections. 2. Improve the potency of the commercially produced vaccine. Dr. Salk believes that, with methods now available, vaccine as potent as that made in his laboratory can be manufactured in massive quantities.

The symposium was presented on Jan. 6 in connection with the national opening of the 1959 March of Dimes campaign. Dr. Salk also reported on the persistence of protective antibodies in subjects vaccinated experimentally almost five years ago. Antibody levels in these children were maintained through 1958 "with only a slight tendency to decline" from levels measured a year ago. Studies supported by The National Foundation on persons who, although recipients of three injections of vaccine, were diagnosed as having paralytic poliomyelitis indicated that illness in about 25 per cent of this group was caused by Coxsackie or ECHO viruses and not by a poliovirus.

Other speakers considered the low degree of immunity in the U.S. population, discussing reasons for the lack of public response to the availability of the vaccine, responsibilities of the physician in this situation, and possible methods of ensuring protection of the entire population against poliomyelitis.

AMA President Dr. Gunnar Gundersen urged communities to find answers to such questions as "Why have only two out of five U.S. citizens had one or more shots and three out of five had none?" and "Why has one third of the entire population under 5 years of age had no vaccine at all, though in 1958 52 per cent of all paralytic cases occurred among children of this preschool age?

"We of the medical profession have long proclaimed ourselves to be public-spirited," Dr. Gundersen continued, "with acknowledged responsibilities for the physical health and well-being of our fellow men. . . . We physicians cannot take the easy way out. Partial immunization is not enough. Local programs must begin immediately so that every one not vaccinated will be inoculated before the next summer epidemic season."

Dr. Thomas Francis, Jr., Chairman of the Department of Epidemiology at the University of Michigan School of Public Health, advocated a code of health describing what every citizen is expected to do to maintain the physical well-being of himself, his family, and his community—just as he is expected to observe housing, electric, plumbing, and sanitary codes. In addition to the health code, Dr. Francis recommended "energetic action" to administer vaccinations to everyone during infancy "regardless of social or economic status" and suggested that a "hard sell" was necessary to reach some portions of the population.

Other speakers included Dr. Alexander Langmuir, Chief of the Epidemiological Branch of the PHS Communicable Disease Center in Atlanta, Ga., who stressed the need for "proper organization and persistent effort" in achieving "universal immunization of the population under 40 years of age"; and Dr. Gordon Brown of the University of Michigan School of Public Health, who, reporting on the extensive laboratory studies made during the 1958 poliomyelitis epidemic in Detroit, said that the vast majority of paralytic cases occurred among those who had received no Salk vaccine at all. Dr. Brown's findings were summarized as follows: 1. The studies suggest that poliomyelitis vaccine may reduce the incidence of nonparalytic as well as paralytic poliomyelitis. 2. Some infections originally diagnosed as nonparalytic poliomyelitis were found to be caused by ECHO 9 virus. ("One may easily conclude from these data that this virus causes more presumably nonparalytic 'poliomyelitis' than does poliomyelitis virus itself," Dr. Brown commented.) 3. Isolation of ECHO 9 virus has even been made from paralytic cases in the Detroit epidemic.

Opportunities for Women in Medicine

FELLOWSHIPS

Industrial Health. Graduate students of industrial health are eligible for three year training fellowships from the Institute of Industrial Health, University of Cincinnati College of Medicine. Offered to students who have completed at least one year of internship, the course of training leads to the degree of Doctor of Science in Industrial Medicine and meets requirements for certification in occupational medicine by the American Board of Preventive Medicine. The first two years of the course are concerned with academic and clinical study in the field; stipends for this period range from $3,000 to $4,000. The fellow also receives compensation for the expense of his final year, spent as a resident in some industrial medicine department or comparable organization. Also offered is a one year course for qualified applicants who may be candidates for the degree of Master of Science. Information is available from the Secretary, Institute of Industrial Health, College of Medicine, Eden and Bethesda Avenues, Cincinnati 19.

Woman's Medical College. Alumnae of the Woman's Medical College of Pennsylvania are eligible to apply for the annual fellowship made possible through the Alumnae Association by funds from the Kate Hurd-Mead legacy. Graduates of the College who wish to engage in some definite study or research project may request application forms for the fellowship, which provides a stipend of $2,400, from Ann Gray Taylor, M.D., Chairman, Kate Hurd-Mead Fellowship Committee, WMC Alumnae Association, 3300 Henry Ave., Philadelphia 29.

GRANTS

Mental Health. Two types of grants have been designed by the National Institute of Mental Health for the psychiatric training of physicians engaged in the practice of medicine other than psychiatry. The first type is available to medical schools, hospitals, clinics, and psychiatric societies for development of training programs in psychiatry. Physicians interested in such training should apply to institutions that have, are are developing, programs in the field. The grants are made to training institutions and do not include direct financial support to individual physicians attending courses, institutes, or seminars wholly or partially supported by the Institute. The second type of grant is offered for support during the psychiatric residency training of physicians in practice who wish to become psychiatrists. Stipends up to $12,000 a year are available. Inquiries about both programs should be addressed to Dr. Seymour D. Vestermark, Chief, Training Branch, National Institute of Mental Health, National Institutes of Health, Bethesda 14, Md.

MEETINGS

Medical Photography. The first Midwestern Sectional Meeting of the Biological Photographic Association will be held April 24-26 at the University of Iowa, Iowa City. The meeting will feature a program of new medical and scientific films and displays of clinical and specimen photography, photomicrography, and photographs of natural science. Photographic specialists in medicine, dentistry, biology, and other sciences will participate. Physicians interested in attending the meeting should contact Mrs. Margaret Ames, Vice-Chairman, 640 E. Armour, Kansas City, Mo.

Medical Writing. The Second Annual Medical Writers' Institute will be held at Rensselaer Polytechnic Institute in Troy, N. Y., June 15-17. The meeting will include workshops, discussion groups, and talks by guest speakers in the field, with most emphasis on the workshop technique. Speakers will be available to provide counseling on medical reports, instructions and circulars, advertising, and medical journalism. Inquiries should be addressed to Prof. Jay R. Gould, Rensselaer Polytechnic Institute, Troy N. Y.

News of Women in Medicine

Dr. Virginia Apgar, Professor of Anesthesiology at the Columbia University College of Physicians and Surgeons, New York City, will participate as a lecturer in the 12th Annual Postgraduate Course in Anesthesiology, to be presented at the University of Kansas School of Medicine in April. Dr. Apgar's subjects are "Neonatal Physiology and the Anesthesiologist," and "Latest Ideas on Infant Resuscitation."

Dr. Rachel Ash has received an achievement award from the Alumnae Association of Woman's Medical College of Pennsylvania, in honor of her distinguished achievement as pediatrician, cardiologist, teacher, and leader in investigative cardiology. Dr. Ash, a member of the graduating class of 1926, retired in April from her post at the College as special lecturer in cardiology. She had served the College in the past as assistant in clinical pediatrics, associate in pediatrics, and clinical assistant professor of pediatrics. A specialist in pediatric cardiology, Dr. Ash has written many articles dealing with rheumatic fever and heart disease in children. She will continue her private practice.

New faculty members at Temple University Medical Center include Catherine Lillie Bacon, Clinical Professor of Psychiatry, and Rosaline R. Joseph, Instructor in Internal Medicine. Recent promotions at the Center include that of Dr. Elsie R. Carrington to the position of associate professor of internal medicine.

The following women physicians from Pennsylvania were recently elected for associateship in the American College of Physicians: E. Cooper Bell, Instructor in Medicine at Woman's Medical College, Philadelphia, and Bernice Elise Durgin, Bellefonte. Dr. Irene Frances Laub, Easton, was elected to fellowship in the College.

Dr. Katharine R. Boucot, Professor of Preventive Medicine and Clinical Professor of Medicine at Woman's Medical College of Pennsylvania, participated as chairman of a panel discussion at the International Symposium on Tuberculosis, held in Philadelphia in November, 1958. The topic of the discussion was Tuberculosis Case-Finding; the Problem in the General Population, Schools, and Hospitals.

Dr. Rachel Darden Davis of Kinston, N.C., has been elected to the North Carolina Legislature and will become the second woman in the Legislature when it convenes early this year. A graduate of the University of North Carolina and Woman's Medical College of Pennsylvania, Dr. Davis' main political interest is in better education at the high school level.

She will continue her 25 year practice in obstetrics and gynecology in Lenoir County, where she was the first woman physician. In addition to her work as representative and physician, Dr. Davis is an amateur gardener and tennis player and enjoys sewing, cooking, reading, walking, and music. She has a 22 year old adopted daughter.

On the program of the One Hundred Eighth Annual Session of the Medical Society of the State of Pennsylvania, held in October, 1958, were Philadelphia physicians Dorothy E. Johnson (Clinical Associate in Pediatrics at Woman's Medical College), Second Vice-President of the Society, and Elizabeth Kirk Rose, responsible for program planning in the field of pediatrics.

Dr. Harriett C. Jones was recently appointed by the Philadelphia Department of Public Health to the District Health Operations Unit, which is responsible for program planning and execution in the 10 health districts in the City. Dr. Jones is one of three recently appointed physicians holding the title of Master of Public Health.

Dr. Esther P. Lovejoy of New York City received special mention in the press release describing publication of the first edition of "Who's Who of American Women." Dr. Lovejoy was described as an "interesting oldster, 89, former president of the American Medical Women's Association, who authored the book, 'Women Doctors of the World,' just last year."

Dr. Mary Hewitt Loveless has been named head of the asthma and allergy program at National Jewish Hospital, Denver. Dr. Loveless was formerly affiliated with the New York Hospital, New York City.

Dr. Alma Dea Morani, Clinical Professor of Surgery at Woman's Medical College of Pennsylvania, stated in an interview with a *Chicago Tribune* reporter in October, 1958, that she believes all plastic surgeons should have some training in art. Dr. Morani said that it would help them to develop discerning judgment, and added that plastic surgeons must be conscious of normal beauty because they try to attain it through surgery. The interview was held in connection with an exhibit of art by members of the American Society of Plastic and Reconstructive Surgery, which was displayed in Chicago.

Dr. Jane Sands Robb, Associate Professor of Pharmacology, State University of New York College of Medicine in Syracuse, left the faculty in September, 1958, after 28 years of service, to become visiting professor of pharmacology at Dartmouth Medical School, Hanover, N.H. Dr. Robb was formerly professor of physiology at Woman's Medical College of Pennsylvania and director of a laboratory of experimental pharmacology at the College. She also organized and became chief of the Cardiac Clinic at Woman's Hospital, Philadelphia.

Dr. Mary Ruth Wester, Instructor in Anesthesiology at Temple University Medical Center, presented a paper on "Manual and Mechanical Control of Ventilation During Cyclopropane Anesthesia" at the AMA meeting in June, 1958.

Aided by a March of Dimes fellowship, Patricia A. Yarvote, a student at Woman's Medical College of Pennsylvania, carried out a statistical study last summer in which she compared several hundred cases of poliomyelitis in patients treated at Monmouth Medical Center in Long Branch, N.J., before the Salk vaccine, with several hundred cases admitted after the vaccine was widely used.

THESE WERE THE FIRST

Dr. Ella Morgan Austin Enlows of Fort Lauderdale, Fla. (born in St. George, W. Va.), graduated from Johns Hopkins University in 1929 and was the first woman on the staff of the Eye, Ear, Nose and Throat Hospital in Washington, D.C.; she was formerly research bacteriologist and serologist with the U. S. National Institute of Health.

Dr. Margaret Fairlie, a graduate of the University of Edinburgh, and Professor of Midwifery and Gynecology, University of St. Andrews, since 1940, was recently elected as a member of the Council of the Royal College of Obstetricians and Gynecologists. She was the only female professor in Scotland. She retired in 1956.

Dr. Orrie R. Moon of Albemarle County, Virginia, graduated in 1857 from the Female Medical College of Pennsylvania (now Woman's Medical College of Pennsylvania) and enlisted as a nurse in the Medical Department of the Confederacy. Only men were permitted to serve as surgeons. Dr. Moon served in the General Hospital at Charlottesville, Va., during the Civil War.

Dr. Romania Bunnell Pratt of Salt Lake City, a graduate in 1877 of the Woman's Medical College of Pennsylvania, was the first woman from Utah to study medicine.

Dr. Frances Ralston Vanzant of Ferris, Texas, graduated from the University of Texas Medical College in 1926, and, after serving as medical director of the University Hospital in Minneapolis, went to Murcia, Spain, as medical chief of the Hospital Paul Vaillant Couturier. Dr. Vanzant retired in 1938.

Dr. Nellie Smith Vernon (1868-1948) graduated in 1901 from the University of Oregon Medical School and moved to Astoria, Ore., in 1903, where she served in the Salvation Army and was appointed health officer for Clatsop County. Dr. Vernon retired in 1946 while living in Portland.

—From the Elizabeth Bass Collection,
Rudolph Matas Medical Library,
Tulane University, New Orleans

Book Notices

Editor's Note: These reviews represent the individual opinions of the reviewers and not necessarily those of the members of the Editorial Board of the JOURNAL.

ESSENTIALS OF DERMATOLOGY. By Norman Tobias, M.D., Fellow, American Academy of Dermatology and Syphilology; Diplomate, American Board of Dermatology and Syphilology; formerly Associate Professor of Dermatology, St. Louis University, Mo.; and Dermatologist, St. Louis State Hospital. Fifth Edition. Pp. 651. Price $8.00. J. B. Lippincott Company, Philadelphia, 1956.

Dr. Tobias' book is a condensed encyclopedia. It is a heroic effort to present all of dermatology in a compact volume. While the common dermatoses are stressed, a great deal of unusual material is included. Each chapter ends with a consideration of the nursing aspects of the previously discussed dermatoses. This is an excellent idea and makes the book of value to nurses and to physicians who must detail treatment to the patient's family. Of course, there are some topics, such as patch testing for contact dermatitis, that could be reworked.

The sections on therapeutics point up again the uniformity of specific treatment and the lack of uniformity of approach to topical therapy.

This book presents Dr. Tobias' own ideas on therapy, as, for example, his feeling against surgery for plantar warts. It is worth-while reading for anyone with an interest in dermatology.

—Thelma G. Warshaw, M.D.

AN ATLAS OF FETAL AND NEONATAL HISTOLOGY. By Marie A. Valdes-Dapena, M.D., Associate Professor of Pathology, Woman's Medical College of Pennsylvania; Assistant Chief in Pathology, Department of Laboratories, Philadelphia General Hospital; and Assistant Medical Examiner, Division of the Medical Examiner, Department of Public Health, City of Philadelphia. Forward by Edith L. Potter, M.D. Pp. 200. Price $11.00. J. B. Lippincott Company, Philadelphia, 1957.

This work fills a need in the reference armamentarium of the pathologist, pediatrician, and medical student by presenting succinctly and cogently material not readily available from the usual reference sources.

Descriptions are clearly written and in the main adequately describe the illustrations. However, in many instances the illustrations fail to depict adequately the features mentioned in the accompanying text, due primarily to the author's use of a magnification too low to present satisfactory detail. It is understood that a number of the low magnifications are required for proper portrayal of baser features; yet, even with this consideration, it is felt that in several instances the illustration is a less than adequate counterpart to the text.

Another criticism is made relative to the arrangement of the descriptive text in relation to the illustrations. As one reads the descriptions it is often necessary to jump back and forth from text to one or more captions on one page and to one or more illustrations on the opposite page. Sometimes it is not readily apparent from the location of the text to which photograph(s) or caption(s) the text refers. Perhaps a different format would make the text and illustrations more easily correlated and reading of the book more enjoyable.

This is a work of sound value, somewhat unique in scope, which presents its technical matter sufficiently well but which, it is believed, would be improved by an adjustment of some of its photographs and by a change of format for easier readability.

—C. M. Plair, M. D.

DRUGS: THEIR NATURE, ACTION AND USE. By Harry Beckman, M.D., Director, Departments of Pharmacology, Marquette University Schools of Medicine and Dentistry, and Consulting Physician, Milwaukee County General Hospital and Columbus Hospital. Pp. 728, with 126 figures. Price $15.00. W. B. Saunders Company, Philadelphia, 1958.

This text, designed primarily for the undergraduate medical student, should also serve as a valuable reference book for practicing physicians. It opens with chapters indicating sources of drugs and basic ideas that point the way to the discovery of new ones, therapeutically useful. There is an interesting chapter on the nature of drug action in terms of what is known of the effect on the molecular structure of living protoplasm.

The greater part of the text is concerned with the practical application of the action and uses of drugs. Some are classified according to the treatment of various systems of the body. Another section deals with the pharmacology of specific symptoms, and another with drugs used as replacement therapy, such as enzymes, hormones, and vitamins. There is a chapter on therapeutic gases, drugs that convey radioactivity, and drugs that are used as diagnostic aids.

With its good system of cross reference, bibliography, and detailed index, this text is easy to use as a reference. For the student, it presents a mass of basic and practical information in a form easily utilized.

—Marion Tyndall, M.D.

ATTENTION AMWA MEMBERS
WE WANT YOUR OPINION

For the purpose of continuous improvement of the JOURNAL OF THE AMERICAN MEDICAL WOMEN'S ASSOCIATION in reading content, it is urgently requested that you spare a few moments to fill in and return this questionnaire.

YOUR RESPONSE TO QUESTIONS BELOW WILL BE MOST HELPFUL

Indicate Your Choice	MORE	LESS
Scientific Articles		
Editorials and Letters		
Special Articles (Features About Women in Medicine and Allied Professions, Historical Articles About Women Physicians)		
News Items and Opportunities		
AMWA Branch News		
"Firsts" and Album of Women		
Book Reviews		

Features to be added or increased _____

_____

Features to be deleted or decreased _____

_____

Indicate your favorite departments or feature _____

_____

Do you read your JOURNAL OF THE AMERICAN MEDICAL WOMEN'S ASSOCIATION?

Every month _____ Frequently _____ Occasionally _____

List medical journals you read in order of interest: Indicate position you would give the JOURNAL OF THE AMERICAN MEDICAL WOMEN'S ASSOCIATION.

1. _____ 4. _____
2. _____ 5. _____
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Please return this page to

Dr. Frieda Baumann, Editor
Journal of the American Medical Women's Association
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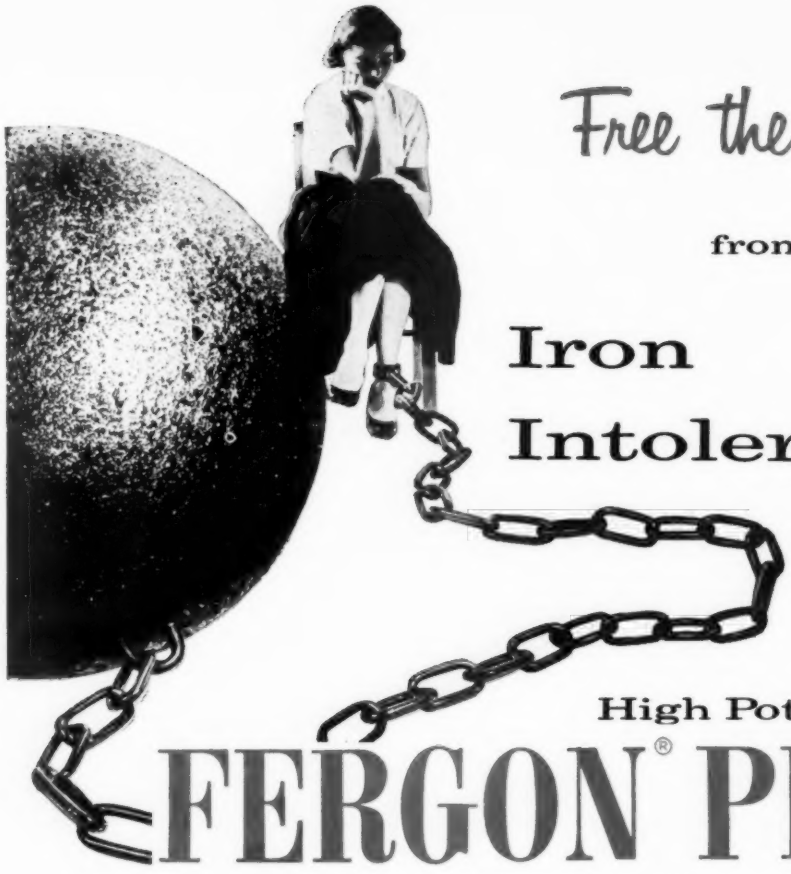

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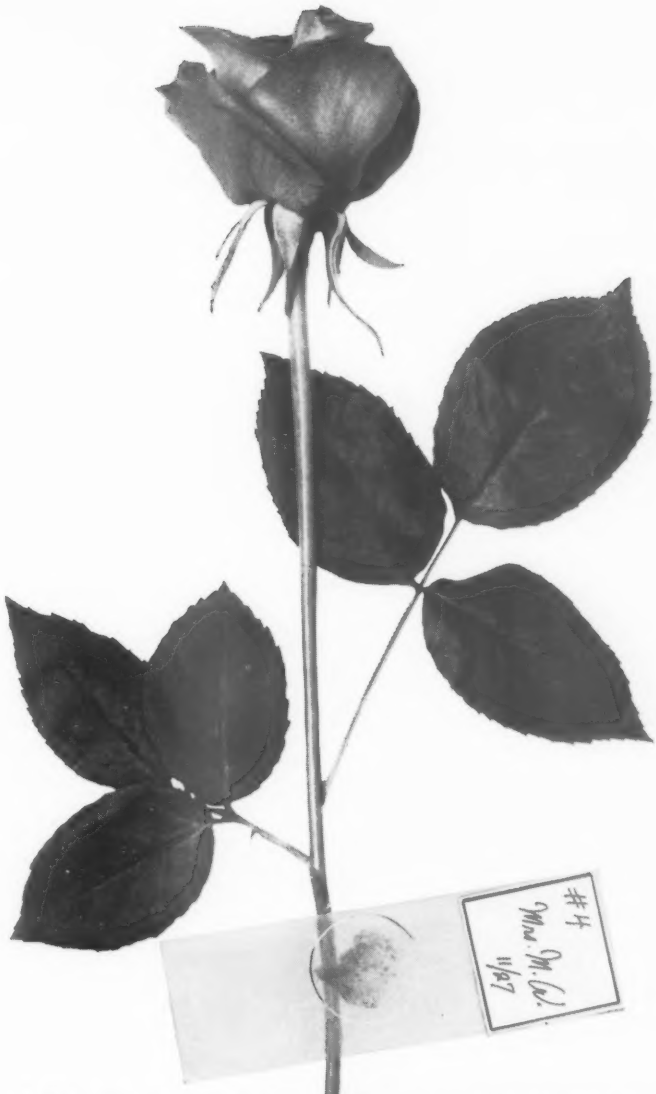

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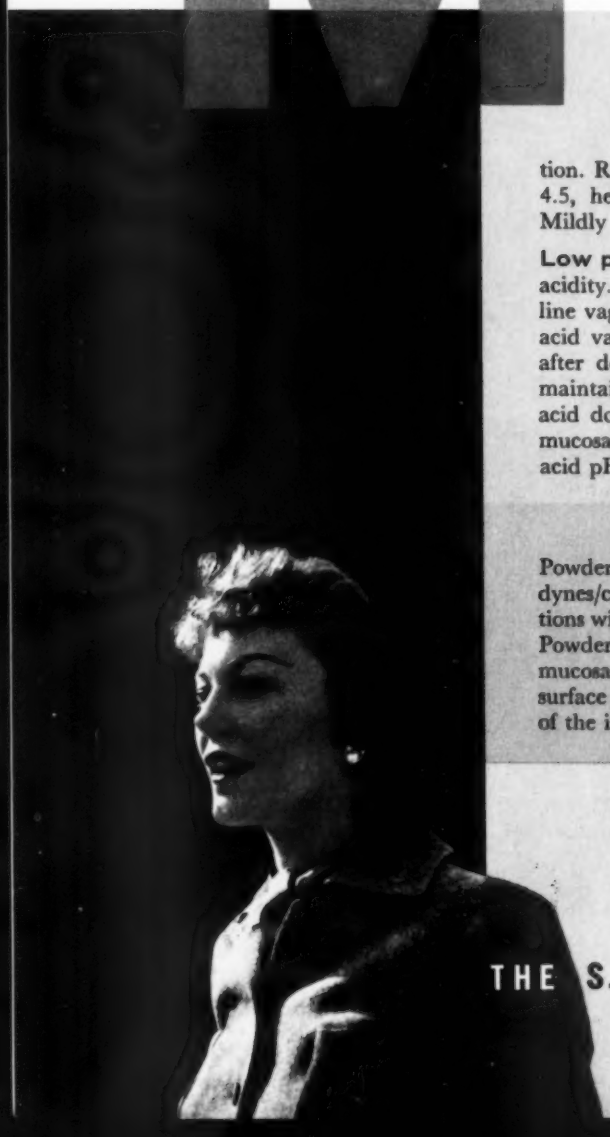

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